TREATMENT OF RESPIRATORY EMERGENCIES INCLUDING BULBAR POLIOMYELITIS

Publication Number 168

AMERICAN LECTURE SERIES

A Monograph in

AMERICAN LECTURES IN OTOLARYNGOLOGY AUDIOLOGY AND BRONCHO ESOPHAGOLOGY

Edited by

NORTON CANFIELD, M.D
Associate Clinical Professor of Otolaryngology
Yale University School of Medicine
New Haven, Connecticut

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RESPIRATORY EMERGENCIES

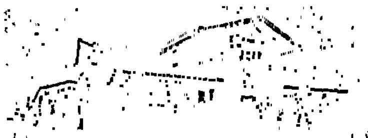
Including

BULBAR POLIOMYELITIS

By

THOMAS C. GALLOWAY, M.D.

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FOREWORD

THE IDEAS and procedures presented in these few pages were developed by discussion and trial in conjunction with a group of alert, thoughtful, and always cooperative colleagues over a period of years. The expression of these views is my own for which I must accept full responsibility. In the treatment of any given case involving respiratory problems there is now seldom important disagreement within the group who form a coordinated team and ask and give consultation freely. Attending and house staff, nurses and technicians, all contributed by suggestion, discussion, and by untiring and attentive care of patients, with the result that an unusual degree of concerted team work has been achieved.

It is not possible to give credit to all of those who merit it. To mention only a few to whom I am indebted, the late Dr. C. A. Aldrich gave especial help in problems of tracheobronchitis. Dr. Jerome Head converted me to the importance of postural drainage. Dr. Martin Seifert directed much of the general care on polio patients and helped develop the treatment here outlined. Dr. Lowell Snorf gave us important consultation and advice. Dr. J. Earl Remlinger gave much aid in respiratory problems. Drs. Louis Sauer, Herbert Lussky, Alvah Newcomb, and Benjamin Rappaport did the same in pediatric cases. Drs. Vernon Turner, Frederick Hiller, Meyer Brown, Gail Soper, John Elsen, John Ballenger, and many others collaborated in working out our problems.

Mrs. Alice Nuel, for many years in charge of the contagious ward, helped greatly to convert clinicians to the

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Respiratory Emergencies

TREATMENT OF RESPIRATORY EMERGENCIES INCLUDING BULBAR POLIOMYELITIS

CHAPTER I

INTRODUCTION

IT IS NOW RECOGNIZED that anoxia, previously unsuspected, is a determining factor in many conditions associated with respiratory obstruction It is also appreciated that secretional obstruction may be one of the most important types of obstruction. In addition, it is well established that marked changes in the pulmonary area may develop secondary to blocking of the airway. These changes may include atelectasis, pneumonitis and pulmonary edema We believe that the timely recognition of these factors and the treatment based upon such recognition have been effective in a variety of conditions.

Methods of care based on such a concept of disease have apparently saved many lives, not only in bulbar poliomyelitis but in acute infectious tracheobronchitis, flame and gas injuries of the respiratory tract and diseases where spasm or secretion may be important. These include tetanus, botulism, cranio-cerebral injuries, vascular accidents and other conditions where similar secondary effects dominate the picture

This discussion is written so that men having to cope with such emergencies may find their decisions easier and may not easily be overwhelmed by a spirit of defeatism in situations which, with understanding, may prove amenable to proper management.

While the approach to this problem has been chiefly clinical it is based on considerable study of the underlying physiopathology. *There is little herein which is original*

The writer has weighed the ideas of known physiology and pathology, often borrowed from apparently unrelated fields, and has accepted or rejected them on the basis of clinical trial. He feels that he has arrived at a rather clear picture of what happens in the conditions to be described and hopes to convince the reader that treatment based on such ideas and experience is logical and effective.

When my first case of bulbar polio was thrust upon me as a full-blown emergency, I had had almost no previous *knowledge of that disease nor any experience with it.* There was not time for discussion, for consultation, or even for detailed analysis. I was conditioned by previous study and experience with tracheobronchitis and allied obstructive disorders of respiration to recognize that there was time only for a decision that must be made in a fraction of a minute. That that decision was right in this particular case at least, was amply justified in the light of the patient's almost miraculous recovery on clearing the airway The experience was repeated in two cases immediately afterward, but my position was assailed by those with preconceptions which generally prevailed at that time As I studied the disease and talked with those who were committed to the older ideas of this disease, my conviction wavered for a time. However, after more experience a belief developed and has remained firm that my first picture was clear and accurate.

I should like for the reader to retrace with me in the pages that follow some of the steps that have led to my present convictions in the care of the conditions discussed, including bulbar poliomyelitis He will then, I think, find our thesis a little more *convincing Some of the by*-ways we explore may appear too diversionary at first but should prove of considerable interest. This apparently devious route should lead us to the goal of better under-

standing of the problems of disturbed respiratory physiology. Such a study, combined with practical experience, seems to me to provide a surer basis than that reached by academic or laboratory consideration.

Many interesting phases of conditions discussed can be merely outlined. For instance, there is much recent investigation of atelectasis which can barely be mentioned. There is also a great ferment of research into the types of poliomyelitis viruses, the method of their transmission and the modes of their action which cannot be touched upon. To avoid these may appear to be oversimplification, but the presentation has advisedly been stripped bare of obscuring details which would only blur the picture. There have also been omitted many controversial issues because their determination in the present state of our knowledge has little bearing on the basic premises of this outline.

Fundamental to our concepts in diseases associated with respiratory obstruction are two main considerations, (1) the secondary effects of respiratory obstruction, and (2) the effects of anoxia The first of these will be developed in relation to acute infectious laryngotracheobronchitis in the second chapter Anoxia will be discussed in the following one Later chapters will develop their application to bulbar poliomyelitis and other conditions associated with respiratory obstruction and secretion

THE MECHANICS OF RESPIRATORY OBSTRUCTION

THIRTY YEARS ago the entity now recognized as acute infectious laryngotracheobronchitis was at about the stage where bulbar poliomyelitis has been until very recent years. Acute tracheobronchitis was considered an overwhelmingly toxic respiratory infection. Relief was sought only in the stage of cyanosis, rapid pulse, and failing respiration, which we now know was the stage of irreversible changes. The reported mortality of the disease was from 50% to 70% (1). Later, even without sulfa drugs and penicillin, Davison (2) was able to report 15 consecutive recoveries in patients requiring tracheotomy. My own experience was almost that good. Few patients are now permitted to succumb to this disease.

In 1924 Baum (3) proposed the idea that obstruction was an important factor in this disease. Developing on that basis and aided by the work of the Jacksons (1), Gittins (4), Richards (5), Orton (6), Holinger (7), Neffson (8), Davidson (2), and many others (9), treatment has taken away most of the dread of this condition. It has been one of the satisfying miracles of medicine to see tiny patients with acute tracheobronchitis, cyanotic and restless or semicomatose, and fluttering on the brink of eternity, become rosy pink and alert or drop off in easy sleep in a short time after proper measures are taken.

To my mind two factors have seemed most important

in this disease First, the mechanical effects secondary to respiratory infection associated with obstruction and increased negative intrathoracic pressure, and second, the effects of hypoxia—or as it is generally denoted, "anoxia" —with associated accumulation of carbon dioxide in the blood stream. The latter will be more fully discussed later. Sepsis in these cases is I believe not of primary importance, and when it is serious is more likely to be due to infection superimposed on atelectasis

In acute obstruction at the laryngeal level, the urgency of impending asphyxia may grant only seconds to save life If the airway is cleared the emergency may be safely past. If however, the obstruction is not critical but is unrecognized and persists, definite serious secondary effects usually develop

The causes of acute respiratory obstruction are many. The most important are outlined below:

1 Simple edema associated with iodides, cardiovascular and renal disease, gout
2. Allergy with edema, spasm and mucus plugs.
3 Acute infections, especially diphtheria, acute laryngotracheobronchitis, abscesses within and without the airway.
4 Sectional obstruction and/or spasm, bulbar poliomyelitis, tetanus, botulism, other central brain disease
5 Positional obstruction due to coma, shock or weakness
6 Chronic infections including tuberculosis, syphilis, leprosy, scleroma and other granulomata
7 Trauma from instruments, caustics, gases, flame, fracture of the larynx and irradiation
8 Tumors, benign and malignant
9 Congenital malformations, webs, tracheal collapse.

10 Foreign bodies.

11. Paralyses, especially bilateral abductor paralysis of the larynx.

As a typical condition acute infectious laryngotracheobronchitis will be discussed in detail. It gives perhaps an extreme picture, but this may be approached by various other conditions, even not primarily infectious First, it occurs usually in very young children where the lax areolar subglottic tissues permit much edema with consequent marked obstruction; and second, associated infection is often grave. This may be secondary rather than primary. Laryngotracheobronchitis has often been overlooked or confused with atypical bronchopneumonia. It does appear however to occur as a primary condition and seems to be a definite clinical entity.

The symptoms are typical. Following an ordinary cold a child may develop hoarseness, croupy cough, restlessness, stridor and then perhaps rapidly increasing obstructive dyspnea. There may follow marked cyanosis and rapid pulse, or the more serious pale cyanosis of exhaustion seen especially after sedatives Obstructive symptoms with retraction, suprasternal, intercostal and substernal usually are outstanding but they may be lacking if opiates have been given or, as pointed out by Gittins (4), if the obstruction is low Marked dehydration often occurs, perhaps because the demand for oxygen is more urgent than that for water These patients cannot take time out from the fight for air long enough to swallow fluids

The exact pathologic process is probably not truly represented by the postmortem findings after tracheotomy and instrumentation *The Jacksons (1) have given a* truer picture by bronchoscopy in the living. Apparently a descending mucositis occurs with first a stage of dry inflammation followed by congestion, edema and exces-

sive secretion. Plugs, crusting and ulceration may occur, especially after tracheotomy without proper humidification of inspired air Schuller (10), after studying the few postmortem specimens he could find not modified by abnormal agonal processes, considered the condition essentially a cellulitis of the mucosa and submucosa with lymphangitis, edema, infiltration of white cells, focal necrosis and vascular changes.

But an interpretation of the disease process in this and other obstructive conditions based on postmortem material gives a poor idea of what really happens. A perversion of the normal processes and functions of the whole respiratory tract apparently occurs For a true picture of this physiopathology there is important evidence in the work of Binger and Moore (11), Coryllos and Birnbaum (12), Kernan and Barach (13), Galbraith and Steinberg (14) and many others These emphasize the far-reaching effects of increased negative intrathoracic pressure, of secondary vascular changes and of damage to the lungs and of superimposed infection

Galbraith's and Steinberg's (14) beautiful experiments showed that bronchial obstruction with associated interference with respiratory movements leads to marked secondary vascular changes especially congestion, stasis, edema and transudation. Kernan and Barach (13) emphasized the importance of these circulatory effects and concluded that "as a result of the continued obstruction and the elevated negative pressure in the chest, blood enters the right chamber of the heart and lungs more freely, but it is retarded in its departure from the lungs into the left heart as a result of the backward suction pressure on the left ventricle as it delivers blood into the extra thoracic aorta The elevated negative pressure within the chest exercises suction on the capillary walls which is the

important factor in the production of intra-alveolar exudate." Auer (15) in 1917 in a study of war gases pointed out that pulmonary edema may be produced by a locally acting mechanism. "Each alveolus which is in connection with a stenosed bronchus or bronchiole will act like a miniature dry cup during inspiration." This initiates or facilitates the passage of a transudate into the alveolar spaces. More will be said about this later in discussing pulmonary edema Air absorption is believed to lead to atelectasis; and rupture of damaged alveolar walls leads to emphysema. Increasingly greater respiratory effort keeps up a vicious cycle.

Other mechanisms have been described for the production of atelectasis and the exact explanations are too controversial to be detailed here. The facts seem to agree, however, with the effects outlined, in respiratory obstruction

It would seem from a study of the above and other work that the important factors in this and similar conditions are the mechanical ones of high obstruction and secondary vascular changes plus superimposed infection. The original infecting organisms have been reported to be chiefly streptococci and H influenzae. At Cook County Children's Hospital in a series carefully studied, green forming streptococci strongly predominated. Bradford and Leahy (16) with a careful plating technique found this same organism predominating with less than 5% of hemolytic streptococci in most cultures The tissue reaction and sticky exudates seen clinically in tracheobronchitis would be in accord with the finding of streptococcus viridans

It may even be that in obstructive disease the picture can be much the same without preliminary infection. With atelectasis, however, there apparently is uniformly superimposed infection and pneumonitis If not relieved

this may go on to focal necrosis or lung abscess and later bronchiectasis as Galbraith and Steinberg (14) found when bronchial obstruction was produced in healthy dogs. In six cases of flame injury where the primary injury was purely traumatic, the clinical and bronchoscopic picture was hardly different from that of acute infectious tracheobronchitis

To sum up the pathogenesis of tracheobronchitis, it seems to me that this description is fairly accurate An infection involves the larynx and subglottic or supraglottic tissues Congestion and edema lead so nearly to obstruction as to raise the negative intrathoracic pressure which causes added congestion, transudation and capillary damage From this as well as from direct infection thick exudate blocks the bronchi or bronchioles whose walls are already swollen As spreading secretion blocks the regional bronchioles, the interlobular tracts are cut off, air in the alveoli becomes absorbed, the dependent portions tend to become atelectatic, and the overlying ones may become emphysematous. Infection and local circulatory changes add to the exudate. Atelectasis and emphysema further embarrass the heart.

Work on experimental pneumonia indicates that a wide variety of effects can occur secondary to blocking Nungester and Jourdonais (17) found it was easy to produce experimental pneumonia in dogs only if they introduced organisms protected from the action of phagocytes by mucin Loeschke (18) concluded from a study of human material that in pneumonia infection was spread chiefly by tidal flow of exudate into open alveoli and bronchioles The resulting condition is apparently simpler, more easily reversible and quite different from the pneumonitis which results from the infection of solid atelectatic lung

In the experiments of Galbraith and Steinberg (14)

already cited and in work on war gases by Winternitz (19) and others, it was shown that when atelectasis and exudate or fluid persist for a period of one or two days *infection is almost invariably* superimposed. The result is pneumonitis or atypical pneumonia and their secondary effects such as focal necrosis, gangrene and bronchiectasis.

In this connection it may be interesting to review our *knowledge of the etiology of bronchiectasis* There seems to be pertinent evidence that it may often result from a single obstructive episode. Perry and King (20) in a careful study of a large group of patients with bronchiectasis found that the process did not progress during *continued observation in more than possibly 6% of their* cases. Goodale (21) established that in at least one third of his patients the condition developed before the age of ten. Anspach's (22) roentgenologic studies of children with bronchiectasis disclosed that on previous films frequently taken several years before, a large proportion had shown the wedge shadows of atelectasis.

It has often been assumed that bronchiectasis was a slowly developing process due most often to the continued feeding of a nasal sinusitis, and countless operations have been done on the sinuses in that belief. It is a matter of observation that the two conditions do frequently coexist. It is suggested, however, that the relationship is usually only coincidental and that the two conditions may have a common etiology which is often allergy. Watson and Kibler (23) believed that 90% of their bronchiectatic patients were allergic They stated also that "the fundamental basis for chronic or recurring sinusitis is commonly an allergic rhinitis" which I believe is true.

Miller and Piness (24) have reported a group of patients in which so-called atypical pneumonia had been

diagnosed, in which obstruction and atelectasis were the essential factors.

Allergy was present in a high percentage of my cases of acute laryngotracheobronchitis. Certainly in an allergic child with edematous mucosa, bronchial spasm and tenacious plugs, anchored perhaps by the mechanism observed by Hilding (25), conditions are ideal for the production of blocking, atelectasis and a sequence following infection that might result in bronchiectasis. I have slides indicating the beginning of this process in an asthmatic child dying of measles encephalitis with atelectasis. There are, of course, many other mechanisms and explanations for the development of atelectasis (26, 27).

Probably there are pathologic conditions similar to those outlined in many cases called atypical pneumonias, especially when these are associated with emphysema, shifting roentgenologic and physical findings and delayed or incomplete resolution Snow and Cassasas (28) reported such a series in an influenza epidemic. They ascribed the findings to obstructive atelectasis and emphysema Holt (29) reported emphysema in a large percentage of autopsies in bronchopneumonia in one epidemic.

It does seem clear, therefore, that a considerable variety of diseases may be secondary to bronchial or higher obstruction which is frequently unrecognized These may include the so-called atypical pneumonias, lung abscess or gangrene, bronchiectasis and bronchostenosis.

This detail has been given to show that the problem in any respiratory obstruction is not simply one of blocked airway and anoxemia, but that grave progressive and irreversible changes may at any time supervene Any treatment then may be unsuccessful.

Rest, fluids and oxygen therapy are successful in relieving most cases of acute infectious tracheobronchitis.

But if these do not give relief, tracheotomy is to be done without delay. It is frequently too long postponed to the point of marked cyanosis, rapid pulse and imminent heart failure. Patients with supraglottic edema, as pointed out by Neffson (8), are likely to be most urgent.

Our treatment has been as outlined below:

1. Rest — physical and mental — so that minimum oxygen is required.
2. No opiates or atropine.
3. Oxygen by catheter, hood or tent.
4. Fluids by mouth and parenterally to relieve dehydration and liquefy secretions.
5. Moisture saturated air, especially after tracheotomy to lessen or prevent crusting.
6. Tracheotomy is not delayed if the foregoing procedures do not soon give relief.
7. Postural drainage and aspiration.
8. Postural irrigation if secretion, plugs or crusts cause blocking.
9. Bronchoscopic removal of crusts or plugs if necessary
10. Expectorants are of questionable value. Water parenterally is the best, but two to four grain doses of iodides seem to help.

The reason for most of the above are obvious. Opiates lower the struggle for air, and the margin is so narrow that unless the patient uses all his power to get oxygen he will soon be overwhelmed Atropine thickens the already sticky secretions and it was observed by an associate at one hospital a few years ago that no child recovered from this disease when atropine had been given.

Oxygen is valuable If it brings any measure of relief and progressive improvement treatment should be expectant and most patients will recover without cannula

tion If, however, the battle proves a losing one, intervention must not be postponed until general and cardiac exhaustion have passed the possibility of recovery, nor until the secondary changes are irreversible Kernan and Barach (13) have emphasized the value of giving oxygen with positive pressure with or without helium.

Indications for tracheotomy in this condition have been.

1. Impending asphyxia
2. Threat of cardiac or general exhaustion
3. Marked and persistent retraction unrelieved by oxygen
4. Restlessness
5. Cyanosis, especially of the gray type, and rapid pulse. These are often signs of imminent heart failure

There has been much discussion of the relative value of intubation and tracheotomy In diphtheria intubation has generally been the treatment of choice It must, however, be done by an expert, and an expert must at any time be available to reinsert the tube which may be coughed out It is better done under direct laryngoscopy with aspiration of secretions and membrane which may even obviate the necessity for it Even here I believe tracheotomy is better.

For all except very temporary obstructions it seems to this writer that tracheotomy is preferable It is more certain for the occasional operator It puts the larynx at rest and allows its earlier return to normal It permits suction through the tube with irrigation Plug and crust removal and bronchoscopy can be easily done through the wound as is often necessary With tracheotomy crust formation is sometimes increased, but that can largely be prevented if the atmosphere is kept at Jackson's (1) dew

point. This is done best by a mechanical humidifier as described by Davison (2). Bronchoscopy for aspiration and inspection of the mucosa should usually precede tracheotomy. With the bronchoscope left in place the patient is usually relieved and the tracheotomy can then be deliberate. When one has done tracheotomies over the bronchoscope, he is not likely by choice to do it otherwise. The Mosher life saver or endotracheal tube are also of considerable value as a preliminary to tracheotomy.

In the condition herein discussed bronchoscopic aspiration is often a necessary and life-saving procedure. As an adjunct postural drainage seems to have been neglected, even though mention is frequently made of the adverse effects of gravity on secretion. Postural treatment is widely used by chest surgeons and internists, but there has been little about it in the medical literature until recent years.

Nelson (30) showed that even in bronchiectasis, in which sputum is viscid and tenacious, proper postural drainage was very effective and resulted in at least temporary cures. He insisted on the patient lying inverted with the hips over a double inclined plane, with the position maintained for days or even weeks with only very brief interruptions Cures by it have been seen in early cases of bronchiectasis in children, and it is usually part of the medical routine in abscess of the lung.

Dr. C A Aldrich (31), who saw many patients at the Children's Memorial Hospital, stated that during one year he treated influenza by postural drainage and did not have one case of pneumonia develop in such patients.

Postural drainage must be such that a real gradient is maintained from the main lower bronchial area to the trachea and larynx According to Morlock (32) this should mean at least 15 degrees elevation of the foot of

the bed, but better as much as the patient will tolerate, which will increase with use. In infants the prone position with the foot of the bed moderately raised provides a much better gradient and it is usually accepted easily.

Having in mind the physiopathology of acute laryngotracheobronchitis it would seem that here postural drainage would be especially valuable. The usual protective mechanism fails Secrections are not moved by cough or tussive squeeze and ciliary action is probably limited Secretion as it is first formed, whether as mucus, transudate, exudate or hemorrhage poured out into the trachea, bronchi or bronchioles, is fluid and will flow some distance along the dependent wall before it forms plugs or crusts. At first ciliary action probably helps. Leonard Hill (33) showed that cilia from the horse's trachea moved material twice as fast in the horizontal as in the vertical position Murphy (34) injected material of the viscosity of secretions found in asphyxia neonatorum into the bronchi of cats and demonstrated that gravity drainage alone could clear the tracheobronchial tree.

If secretion can flow to the areas of greater cough reflex, tussive squeeze and cough will be much more effective If any cough, squeeze, or ciliary action is left the help of gravity will be important. If secretion stagnates it should then be as a spread dependent layer and not as an occluding mass lying in a bronchial well If plugs or crusts form they will be nearer the outlet as we have observed bronchoscopically after instituting postural drainage These will be more easily expelled by cough or will be more accessible to removal by aspiration or bronchoscopy

Lavage of the respiratory tract has been proved feasible by Stitt (35) and by Minter (36), the latter using as much as 400 cc of fluid at a time in adults Gittins (4) in

tracheobronchitis used epinephrine and physiologic solution of sodium chloride in small amounts, aspirating it afterward. Richards (5) showed that a solution of sodium bicarbonate tends to soften and dissolve plugs.

Wetting agents such as sodium lauryl sulfate have been advocated to prevent crusting and seem to be of real value. The quantity should be sharply limited as foaming may occur. In one case of bulbar poliomyelitis I felt that this contributed to an unfavorable outcome

Penicillin in normal saline has been used for its local antibiotic effect to soften plugs and to thin secretion.

If one is to use fluid it would seem that after it is instilled the inverted posture should be used to guarantee its return and also to aid in the movement of secretion and plugs toward the tracheotomy opening. In our cases 4 to 15 cc. of from 3 to 5 per cent of warm solution of sodium bicarbonate at a time have been dropped into the tracheotomy tube during inspiration, usually with the gratifying result that vigorous coughing presumably churned the solution over crusts and plugs. This procedure was carried out with the head elevated and the child turned toward the side on which the stethoscope indicated the greatest blocking The head was then lowered and suction was used by a catheter through the tracheotomy tube Physiologic solution of sodium chloride or Ringer's solution followed to wash out the sodium bicarbonate. Intermittently when there was much blocking epinephrine solution 1 10,000 was used (Stronger solutions are known to produce undesirable secondary congestion in the nose) This treatment has proved of much value in recent cases, although it cannot entirely supplant bronchoscopic aspiration and forceps removal of crusts It proved especially valuable in cases in which even a 3.5 mm bronchoscope could not be passed A

capable house officer can be trusted to use postural irrigation and save the laryngolist constant attendance and the patient tiring bronchoscopies.

This condition discussed in detail varies from other obstructive respiratory disease because it occurs in younger patients and because there is associated infection from the beginning But it seems to me that the condition is primarily mechanical and that as in experimental work cited, if obstruction is marked and prolonged, infection is likely to be superimposed The severity of the secondary effects will vary inversely with the age of the patient and is, of course, much less likely to be so serious in the older child or adult.

For instance, in some cases of flame injury to the respiratory tract which I have seen, the end picture in addition to first or second degree burns was hardly distinguishable from that of acute infectious tracheobronchitis The treatment probably should be almost the same Such cases probably are often overlooked because of associated serious injury Livingston (37) has reported three of these cases in which the symptoms had at first been ascribed to obvious carbon monoxide poisoning. He noted obstructive symptoms and performed bronchoscopies. First and second degree burns were found with intense congestion, edema and exudate in the trachea. Tracheotomy was done with recovery of all three patients. The other three cases I saw were due to inhaling flaming gas in auto accidents and in these the picture was clouded by head injury

In the Coconut Grove disaster "irritation from hot and noxious gases" apparently was responsible for many of the bad effects (38)

Where the mechanical factors of obstruction are chiefly concerned the following conclusions seem valid

1. In laryngeal obstruction relief of asphyxia is the most immediate problem.
2. Persisting obstruction leads to marked changes secondary to increased negative intrathoracic pressure. These are congestion, edema, exudate or hemorrhage, atelectasis and emphysema.
3. Superimposed infection may lead to atypical pneumonia, pneumonitis, lung abscess or bronchiectasis.
4. Intervention must not be delayed until these irreversible changes are established.
5. Postural drainage and irrigation are valuable aids after tracheotomy.

CHAPTER III

ANOXIA IN CLINICAL STATES

WHILE IN GENERAL the academic and theoretical aspects of oxygen want are well known, it does not seem equally true that the importance and serious implications of anoxia are always appreciated, especially under the stress of emergency. A previous discussion by the author (39) was presented in somewhat greater detail to show that some degree of anoxia frequently is unsuspected and untreated, to indicate that even brief periods of hypoxia or anoxia may cause serious damage, to point out some conditions responsible for anoxia, to review the signs and symptoms which may indicate its development, and to present measures for its prevention and treatment

It has been difficult to establish the true histologic picture of damage from anoxia because the most notable injury is to brain tissue in which similar changes occur in association with other acute circulatory and degenerative disturbances Also anoxia is frequently associated in man with other grave conditions such as anesthetic depression and septic, degenerative or circulatory disease In addition it requires a survival time of from 24 to 48 hours for demonstrable histologic changes to develop in brain tissue after injury from anoxia. Agonal and postmortem changes in this tissue are also hard to prevent or evaluate

Windle and Becker (40) have given a clear-cut demonstration of the effects of oxygen want in guinea pig

embryos at term. The uterine circulation was interrupted until asphyxia occurred, the animals were resuscitated, allowed to survive for a time and later killed with the tissue fixed in situ. Although it is accepted that brain tissue of the young is less sensitive to anoxia than that of adults, all animals in whom the circulation was interrupted for eight minutes or more showed neurologic symptoms and pathologic changes. The symptoms included incoordination, ataxia, spasticity, tremors, convulsive seizures, diminished learning ability and even decerebrate states Histologically, in 65% of the animals there occurred edema, chromatolysis, necrosis, capillary hemorrhages, glial proliferation and loss of brain cells with changes varying from slight atrophy to marked sclerosis

Morrison (11) demonstrated similar profound changes in the brain of dogs and guinea pigs subjected to repeated short periods of anoxia. The critical level seemed to be a reduction from the normal 20, to 12 or 13 volumes per cent of oxygen in inspired air following repeated exposures of 25 minutes. For greater degrees of anoxia the time of dangerous exposure seemed to be correspondingly lessened He found in the monkey that a single exposure to a simulated altitude of 32,000 feet for 25 minutes was capable of producing extensive laminar necrosis of the cortex. Frank necrosis occurred usually only after episodes of anoxia sufficiently severe to produce cessation of respiration The frontal lobe was most often involved, the temporal lobe least often The cerebellum was more frequently involved than the basal ganglia, and the medulla least These experiments and many others from the laboratory show definitely the grave and lasting effects of periods of anoxia even as short as *three to five* minutes (12)

Yet these probably do not represent the extreme picture since it is very difficult to exclude such reservoirs of oxygen supply as the placenta and the lungs, as in Windle's experiments, or to completely exclude cerebral circulation through collaterals even after ligation of the carotid and vertebral arteries.

In clinical conditions it has been somewhat difficult to establish the picture of anoxia The studies by Courville (43) of material obtained after death from nitrous oxide anesthesia are classical and his conclusions that these represent the effects of anoxia are quite generally accepted He lists such changes as sclerosis of scattered pyramidal cells, patchy necrosis, degeneration of limited portions of the cortex and lesions in the lenticular nucleus Degenerated areas tend to become confluent and to bring about necrosis of the various laminae. He states that only when the patient survived a sufficient time, usually 24 to 48 hours, are histological changes found, and in the more fulminating cases these were not demonstrable. He believes irreversible changes may occur after three to eight minutes of anoxia

Many authors have shown similar changes, as for instance Schreiber (44) in asphyxia neonatorum and Hartman (45) following surgical operations Since it is not beyond controversy that the effects of carbon monoxide poisoning are due solely to oxygen want the many reports on this disease are not cited

The data so far presented are related to gross laboratory and clinical demonstrations But it is hardly to be doubted that in patients surviving there are important changes beside those effecting immediate circulatory collapse and general depression Courville (46) lists among sequelae of oxygen want, twitchings, emotional flattening or psychic aberrations He believes that the condition of

lapse (53, 55, 56). All of these have been reported as symptoms of bulbar poliomyelitis

Blalock, Harrison and Wilson (53) demonstrated in experimental respiratory obstruction that carbon dioxide

PHYSIOLOGY OF RESPIRATORY OBSTRUCTION

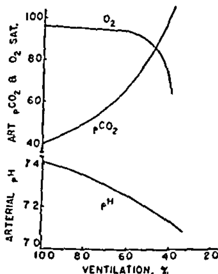


Figure 1 Upper scale represents arterial O_2 in per cent saturation and below it CO_2 tension in mm of Hg Lower scale shows arterial pH and the horizontal scale the adequacy of pulmonary ventilation. (Reproduced with permission from J P. Gray The Physiology of Respiratory Obstruction *Ann Otol, Rhin & Laryng*, 59 72, March, 1950)

may reach dangerous levels in the arterial blood even while oxygen saturation is still normal. This is shown graphically in the illustration from Gray (54), Fig. 1.

With moderate hypoventilation at 80% of normal the oxygen saturation is not detectibly affected, but the CO_2 tension rises to 50 mm of Hg and pH falls to 7 35 which

are definitely beyond the normal range. At 50% of normal ventilation the O_2 saturation falls to about 88%, equivalent to an altitude of less than 10,000 feet and readily tolerated The CO_2 tension however, has reached 80 mm. Hg. which approaches the narcotic level for this gas. The pH has then fallen to 7.2 which is a real acidosis. Slightly further increase in CO_2 may be lethal.

The facts cited make it increasingly clear that the administration of CO_2 to stimulate the respiratory center is not indicated in asphyctic states For the same reasons we have felt that the oximeter, which does not measure CO_2, could not be relied on in place of close clinical observation. This should indicate also that mere administration of oxygen may add little Alveoli may be blocked by aspirated or inflammatory secretion or by transudate or exudate. Damage to respiratory cells by anoxia or edema may suspend their function The first essential is a cleared airway to allow not only absorption of oxygen but what is even more important the elimination of carbon dioxide

The symptoms of anoxia are important to have in mind, especially in dealing with conditions associated with similar manifestations Close analysis will disclose that in many of these it is the anoxia itself which causes the disturbances which may have been ascribed to sepsis, decompensation, shock, virus infection and the like.

Mental symptoms occur early As Barcroft (52) points out, these may resemble those of acute alcoholism and may vary with the patient's temperament and surroundings There may even be preliminary exhilaration and excessive confidence in faulty judgments such as aviators have at high altitude More common, however, are depression, confusion, disorientation, irrationality, unresponsiveness, lethargy and finally coma. Antagonism and combativeness often occur This was found in a consider-

able percentage of our bulbar poliomyelitis cases and has been important because it has made necessary more active measures, such as tracheotomy, which could have been avoided if the patient had cooperated in simple aspiration. It was interesting in reviewing the nurses' records of bulbar poliomyelitis cases to find that confusion and irritability had been frequently noted before respiratory difficulty was yet suspected by the physician.

Restlessness is an important early symptom both because the active patient increases his oxygen need and also because the unwary are too often tempted to attempt its relief by sedation. This may be followed by complete surrender by an overburdened respiratory mechanism

Dyspnea is usually an early symptom but may not be recognized easily in an exhausted patient or in one who has had sedatives.

Cyanosis may be very difficult to recognize and evaluate. It may be a late symptom of oxygen need since it may not occur while there is compensation by increased rate or depth of respiration, and increased rate and stroke output of the heart If obstruction develops slowly, cyanosis may be a late sign I believe that if severe cyanosis develops in conditions of less acute onset, we are likely to have overwaited the indication for tracheotomy Cyanosis may be absent in anemia since it depends on an absolute amount of reduced hemoglobin in the capillaries. It may not be evident in the type of tracheobronchitis described by Gittins (1) which may have only a gray cyanosis and few obstructive signs. This may be, however, simply part of a shock picture.

The effect of anoxia upon the heart is apparently not so great as that of the usually associated carbon dioxide excess. There will frequently be an early increase in rate and stroke output with later rise and finally serious dis

turbance of rhythm. It is to be remembered that a rapid pulse of poor quality is a sign of impending heart failure which again may mean that we have waited too long to intervene.

The immediately adverse effect of anoxia on the respiratory center, on vasomotor control, on the adrenal glands, cardiac muscles and other organs (55, 56, 57, 58), and its part in the increase of permeability of the capillaries of the lung bed (59) must be recognized and combatted early.

Any condition in itself depressing may make a patient unduly sensitive to slight degrees of oxygen want and such patients must be closely watched. Shock is likely to be associated with a stagnant anoxia due to pooling of the blood in the splanchnic area Not only may oxygen administration then be of value, but it is important that the airway be kept clear and the patient, especially if unconscious, be placed in the best position to breathe easily Military surgeons report that unconscious shocked casualties were frequently greatly helped by turning the patients to the prone position with the face to the side.

Anoxia in tracheobronchitis and respiratory obstruction has been considered chiefly as a phase of terminal asphyxia. It should rather have been pictured like a wolf at the heels of a wounded animal, nagging, harrying and tiring until it closes in on its weary prey in one final cruel slashing attack But from the moment in respiratory obstruction that any degree of oxygen want develops it begins to operate adversely and unless relieved it may be largely responsible for an unfavorable outcome It produces restlessness with further activity and increased oxygen consumption. It and the accompanying carbon dioxide accumulation lead to increased respiratory effort, increased negative thoracic pressure, increased conges-

tion, exudate and even capillary hemorrhage, increased lymph flow and increased capillary permeability shown by Drinker (59) to follow anoxia. This exudate leads to still further blocking of the bronchioles and the lung bed, with perhaps atelectasis and emphysema and deepening oxygen want. There is evidence also that vasomotor tone is seriously impaired by depression of function of the suprarenal glands by anoxia or by its effect on the autonomic system (58).

If hypoxia has persisted for some time or there is severe anoxia of even short duration, the heart and respiratory muscles cease to function efficiently due to oxygen want. The fight may quickly near its end. In most conditions associated with anoxia the physio pathologic disturbance must be visualized from the outset if it is not to become irreversible and if tragic sequelae are to be prevented.

Oxygen administration, rest, postural drainage, aspiration of secretion, parenteral fluids and other measures will relieve most cases of tracheobronchitis and other types of respiratory obstruction But undue complacency should not delay the usually certain and relatively safe relief by tracheotomy

This discussion of the effects of anoxia should make the essential picture somewhat more clear in the diseases we are considering Especially is this so in tracheobronchitis, bulbar poliomyelitis and other secretional or spasmodic disturbances. It should help to separate in our minds the effect of infection or sepsis from those of anoxia and carbon dioxide excess But most important it should allow us to concentrate our efforts where the enemy is vulnerable and where treatment may be really effective.

CHAPTER IV

ACUTE BULBAR POLIOMYELITIS

THERE ARE two extreme concepts of the disease of bulbar poliomyelitis One which prevailed up to about 15 years ago was that of a fulminating virus infection which often overwhelmed the central nervous system, especially the brain stem and vital centers The other concept is that it is more often chiefly a problem in respiratory obstruction Both factors probably contribute to the clinical picture to a varying degree.

CONCEPTS OF THE DISEASE

My first experience with bulbar poliomyelitis came as an acute and dramatic emergency The patient had quickly passed from restlessness and disorientation to deep coma An alert supervisory nurse who had assisted in the successful care of many patients with acute tracheobronchitis came upon the scene as internes were desperately trying to save this man. She suggested that this was a respiratory emergency and advised that a laryngologist be called I found the patient deeply cyanotic and almost pulseless Viscid secretion overflowed from the nose and mouth and was seen by direct laryngoscopy to cover the larynx The patient stopped breathing for an estimated three minutes

There was no time for discussion or analysis In the light of my previous experience with obstructive disorders of respiration this seemed at a glance to fall in the same category A tracheotomy was hastily performed, the

trachea was aspirated, manual artificial respiration started and the patient placed in a respirator. In 30 minutes the pulse and color were good and the patient was rational In 45 minutes he was demanding to be taken from the respirator.

It seemed impossible to assume that relief of any purely infectious central process could account for such rapid recovery. In this and other respects it fitted into the picture of obstruction, with anoxia. Similar experiences followed in two other cases shortly afterward, although one died before his tracheobronchial tree could be cleared of the thick secretion which filled it.

I had had no previous experience with acute bulbar poliomyelitis, but had seen many cases of acute tracheobronchitis at Cook County Hospital and in my own practice. I was at first considerably confused with ideas then current, but with increased experience I have firmly continued to believe that relief of secretional obstruction and its associated effects are the most important elements in recovery from bulbar poliomyelitis. This conviction has been strengthened by the support given to it by Dr. Martin Seifert and other colleagues at the Evanston Hospital It must be admitted in fairness that some of my associates have had reservations against complete acceptance of these ideas However, most of the doctors who have critically observed the care advocated have been converted to its use One member of the house staff had opposed as strongly as an interne could the performance of a tracheotomy in the first case he had seen After he had followed three such cases he then said, "If I get bulbar poliomyelitis I want a tracheotomy done right off!"—a more extreme position than I have yet taken

On the basis of my own experience with other respiratory difficulties postural drainage, aspiration, and trache

otomy under proper indications appeared to be logical measures in the treatment of bulbar poliomyelitis Apparently a good many other men have reached the same conclusions by other approaches I later found that Durand (60) in 1929 had advocated postural drainage "since many men must have noted that these patients tend to drown in their own secretions" Wilson (61) had suggested the guarded use of tracheotomy in this disease in 1931 although there had been few reports of its employment in the years immediately following.

In 1943 the writer (62) presented his ideas of the disease, and treatment based on that concept of the disease, was outlined. Then and subsequently the writer (63, 64) and his colleagues attempted to visualize for clinicians the nature of the processes in this disease and the underlying patho-physiology It was emphasized that a prime requisite was to clear the airway and keep it so; that postural drainage and continuous aspiration greatly helped to attain this end, that tracheotomy, far from being a radical and dangerous procedure, in itself carried little risk; and that when indicated it should be done early and not withheld as a last resort until pathologic changes had become irreversible. It was further pointed out that the use of the respirator might be very valuable with these patients when they were exhausted or the respiratory center was depressed by anoxia and hypercapnia. It was also demonstrated that it was not dangerous to use the respirator if the airway was kept clear by performing tracheotomy to bypass secretions These ideas will be elaborated in the following pages

In recent years great attention has been paid to the respiratory problem in poliomyelitis. Many reports have appeared of the widespread and successful use of tracheotomy The reports on a large scale by Priest, Boies and

Goltz (65), by Miller and Buck (66) and many others seem to have established that the combating of secretional obstruction is a sound basis for care in this disease.

The experience of the past five years at the Evanston Hospital is given in Table I.

TABLE I

MORTALITY IN BULBAR POLIOMYELITIS

Place	Year	Total Polio	Bulbar and Bulbo-spinal Cases	Bulbar and Bulbo-spinal Deaths	Bulbar and Bulbo-spinal Rate
Evanston Hospital	1947-48	127	15	0	0
Chicago Residents	1948	312	49	19	38.7%
Illinois Outside Chicago	1947-48	1212	103	39	38.8%
Evanston Hospital	1947-52*	633	129	24	18.6%
Illinois Outside Chicago	*1947-52*	*5018*	*722*	*277*	*38.3%*

* In the last two years a disproportionate share were more serious cases brought from outlying areas and hospitals

It is admitted that the severity and the symptoms vary in the different types of the disease, of which at least three main classifications seem to have been established. Our fifteen consecutive recoveries were seen early under ideal conditions by a compact group well agreed on the principles of respiratory obstruction. We had complete control of our cases and intervened when we considered it necessary. Most of the patients were young, and the strain of the virus may have been mild, yet we were sure six of them would have died without tracheotomy. Another group were vigorous adults who came from greater distances, were treated after greater deliberation and did less well. They were young adults and had, in part, an *ascending spinal infection and probably did have greater* central nervous system involvement.

We have already discussed anoxia and the secondary effects of respiratory obstruction Bulbar poliomyelitis has many features in common with other respiratory obstructions Some of these are. the identity of many of the symptoms with those of anoxia and carbon dioxide excess, the rapid worsening at a critical level after a period of relatively moderate difficulty, the usually rapid amelioration of such symptoms when the airway is cleared before the stage of irreversibility is reached, and the large percentage of cures based on treatment directed to the relief of obstruction

TYPES OF RESPIRATORY DIFFICULTY

We classify respiratory difficulty in acute poliomyelitis clinically as of four types With the first three there is general agreement

1 *Spinal.* In the spinal type of respiratory difficulty the lesions are in the cervical and upper thoracic segments of the spinal cord with varying paralysis of the intercostal muscles and the diaphragm Patients with this type do well in the respirator and fall easily into rhythm with it.

2 *Bulbar* In this type there is interference with the swallowing mechanism and resultant accumulation of secretion, food and fluids, and vomitus in or over the airway If complications do not occur patients usually get over this difficulty in five or 10 days

3 *Bulbar-spinal* This is a combination of the two foregoing types

4 *Other disturbances* in rhythm and depth of respiration Theoretically these could result from a virus effect on the respiratory centers with a fulminating course We made no such diagnoses in the 15 consecutive cases already mentioned We considered the clinical effects observed in

so-called poliomyelitic encephalitis to be due usually to peripheral secretional obstruction plus the effect of anoxia and carbon dioxide excess on the medulla and other centers. Most patients were relieved rather promptly by the measures directed at relief of the obstruction and anoxia.

The picture of the so-called arrhythmias is confusing. One often sees patients with shallow, interrupted, cogwheel and wavelike respiratory movements. At times the intercostal muscles and diaphragm almost appear to oppose each other. We have seen such arrhythmias controlled (with serious results) for a time by thiopental sodium, which would hardly be the case if the arrhythmia were due to central depression.

A simpler explanation of this type of respiration is that it comes from a reflex or voluntary conflict between respiratory demand and the protective mechanism which help to keep the patient from drowning in his own secretions. If the reader would try to imagine what would happen to himself if he were in the position of the patient with poliomyelitis, unable to swallow, with secretion lying in and over his airway, struggling for air, yet at the same time fighting to keep fluid from flooding his lungs, he might visualize the predicament of some of these patients and more easily account for the type of respiration. In my experience these irregularities usually disappear if free respiration is provided without delay.

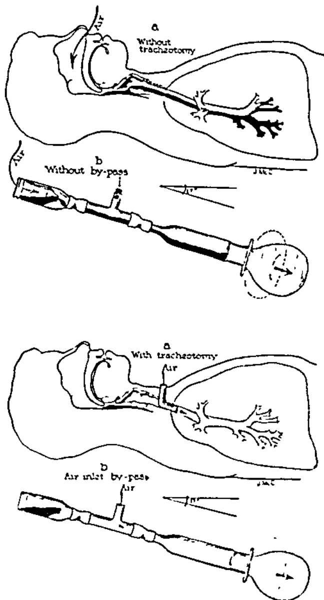
We believe, then, that the primary difficulty in bulbar poliomyelitis is difficulty with the swallowing mechanism resulting in accumulation of secretion, food, fluids and vomitus in the airway with concomitant anoxia. It is to be recalled that there is normally secreted from 1,000 to 1,500 cc. of saliva per day which is likely to be increased in nervous disease and with nausea (67). To this must be

added inflammatory secretion, food or fluid injudiciously given, and vomitus We have recovered by suction from the pharynx in 24 hours over 2,000 cc. of fluid above any added for irrigation.

The usual mechanisms of expulsion of the fluid from the airway are ineffective in these patients Cough does not operate effectively except where perfect postural drainage allows no fluid to accumulate at the level of the larynx or below it, since to get enough air for the expulsive force a deep quick inspiration would be necessary This results in aspiration of the overlying fluid as shown in Figure 2. Both voluntary and reflex control tend to inhibit such inspiration Cough may be difficult also because the medullary center is depressed even early, we believe, from anoxia and excess carbon dioxide Therefore, unless secretion is continuously removed by treatment it will lead to anoxia by preventing ingress of air, by blocking bronchioles and by flooding and secondary damage to the respiratory area of the lungs The best evidence for the importance of anoxia is that the symptoms are usually so promptly relieved when the airway is cleared.

The chief objection to the ideas here presented seem to depend upon histologic evidence that in bulbar poliomyelitis there is widespread damage to cells in the brain stem and medullary centers as well as other parts of the brain and spinal cord

It has been assumed that derangement of function should correspond closely to the areas of microscopically indicated involvement and that symptoms are to be explained on that basis It has been shown that the reticular substance may show marked abnormality in poliomyelitis (68, 69) It seems probable from the work of Wang and Ranson (70) that this area is related to respiration and to vasomotor regulation The inference might then be made


Air
a
Without tracheotomy
b
Without by-pass
Air
a
With tracheotomy
Air
b
Air inlet by-pass
Air

that disturbances of respiratory rhythm and of vasomotor tonus might be explained on that basis From the clinical viewpoint it does not appear to be that easy Pathologic considerations also raise considerable doubt

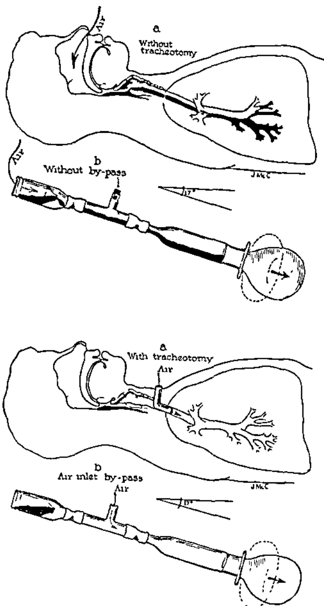
Bodian (71) after intensive study from 24 fatal human cases of poliomyelitis and from studies on experimental monkeys, concluded that "the use of pathologic material from such fatal cases in the correlation of symptomatologic lesions is dangerous " He further said, "As far as the pathologist is concerned all cases of poliomyelitis are encephalitic." He adds, "Apparently severe destructive lesions are necessary to produce dysfunction at the clinical level " It is also important to emphasize," said Bodian (72), "that although lesions appear in certain centers in the central nervous system, symptoms attributable to such injury need not result Injury must reach a certain threshold of severity, varying with the margin of safety of each center before a clinical effect is observed " He also saw no evidence in this material that edema plays a conspicuous role in paralysis

Neuberger (73) and others have suggested that some of the brain damage is due to hemorrhage and anoxia. Courville (74) states that "alterations in the nerve cells are due to the combined effect of the specific virus and of the

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Figure 2 Model showing the effect when secretion lies in the upper airway When by pass is open fluid is not drawn in by voluntary or mechanical respiration as shown in lower diagram upper diagram shows the effect without by pass or tracheotomy With moderately viscid fluid, intravenous tube connector and asepto syringe, model acts as shown (As indicated, the process in patient with bulbar polio probably approximates this without and with tracheotomy) (Modified from illustration in *Arch Otolaryng*, *46* 125, Aug, 1947, with permission)



Air
a
Without tracheotomy
Air
b
Without by-pass
JMcC
a
With tracheotomy
Air
b
Air inlet by-pass
Air
JMcC

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holds true in human drowning. It is probable that in many cases if flooding occurs, it is in the terminal asphyxial depression.

The experiments of Coryllos (77) on asphyxia by submersion are of interest in comparison with secretional obstruction Dogs, he found, succumbed after four or five minutes of submersion. He divided the process into four phases each lasting about one minute. In the first phase there was apnea caused by reflex closure of the glottis. *Rise in blood pressure also occurred which, he states, "is a constant phenomenon in all forms of asphyxia."* Stage two in the second minute is characterized by labored respiratory movements and continuation of the rise in blood pressure (This might parallel the rise seen often in bulbar polio, ascribed by some, and I believe rightly so, to carbon dioxide accumulation) At the end of this phase consciousness disappears and tonic and clonic convulsions appear. In the third phase respiration is arrested, blood pressure drops and reflexes and muscle tonus disappear—including loss of corneal and glottic reflexes The fourth phase is characterized by progressing weakness and arrest of cardiac contraction

Prognosis varied greatly in each phase. If submersion was interrupted before the middle of the third phase immediate and spontaneous resuscitation always followed. At the end of this phase even artificial resuscitation was difficult or impossible In the late third stage, as the heart *began to fail, there was a period of only 10 or 15 seconds* when resuscitation might succeed Coryllos' statement that "seconds count in asphyxia and often determine the life or death of the patient" should spur to decision any one who temporizes with any form of respiratory obstruction

In some of our patients with bulbar poliomyelitis with

asphyxtic symptoms we found almost no fluid on opening the trachea although the hypopharynx was flooded. This indicates that spasm was most important in these cases. Kelleher (79) reports that a patient with poliomyelitis choked on a single teaspoonful of water and died before relief could be given. One of our patients apparently convalescing well was given cracked ice and choked, became cyanotic and died in a short time. One might well ascribe this to strangulation by spasm.

It has seemed to me that spasm is much more likely to be a factor than obstructive median paralysis of the vocal cords. We are not sure we have even seen much paralysis in poliomyelitis. Furstenberg (80) has pointed out that supranuclear bilateral paralysis is extremely unlikely and nuclear or infranuclear paralysis of the cords is flaccid and not spastic. Abductor paralysis due to recurrent laryngeal injury is unlikely in this disease. Sjoberg (81) postulates, largely from x-ray studies in poliomyelitis, that a combination of spasm and paralysis of the muscles of the tongue, jaw and pharynx with the head extended blocks the airway.

While some of our cases with respiratory distress relieved by tracheotomy had dry lower airways, from most of them varying amounts of secretion could be aspirated through the trachea. As previously shown this could come in part as the result of increased negative intrathoracic pressure caused by the obstruction. It would be more likely to come with more slowly developing obstruction, I believe, due to a combination of habituation to secretion or suction tubes in the hypopharynx, loss of the swallowing reflex, inability to cough and slowly developing central depression. Even in these patients spasm may precipitate a crisis. Spasm is especially important in other conditions to be discussed in another chapter. Strobel

and Canfield (82) suggest that a partial paralysis destroys the protective function of the larynx. This could be so, especially if the paralysis were of the flaccid type.

The comparison to drowning is useful since in poliomyelitis the time may be almost as short and the moment for favorable action may as quickly pass. Ignorance or indecision and inertia may hold to stupid inaction a man who would without hesitation leap into a raging torrent to save a child in hardly graver danger.

TREATMENT

Treatment of these patients may be relatively simple or hopelessly involved. If undertaken early, decisively and with a clear idea of the problem, it usually offers little difficulty although it requires meticulous and unremitting care. If delayed by lack of early opportunity or indecision, it may require the utmost in experience, skill, team work and equipment. These cases should have the services of a well integrated team of general clinician, internist or pediatrician, anesthesiologist, neurologist and laryngologist The last is an important member of the group. He should see these cases early and frequently to be able to note any unfavorable course. Decisions for or against tracheotomy should not be made by men who have not had experience with respiratory obstruction without consulting the laryngologist.

POSTURAL DRAINAGE AND ASPIRATION

Postural drainage and continuous suction, best obtainable by a Venturi type pump or central vacuum tank are in our opinion the most important steps early in treatment When the patient first gives evidence of pharyngeal paralysis by muffled voice and inability to swallow secretions, even though he can apparently cough or push them

out of his mouth, he should be placed with the feet elevated and the neck extended, or with face down if a child, so fluid may accumulate in a pool at the naso

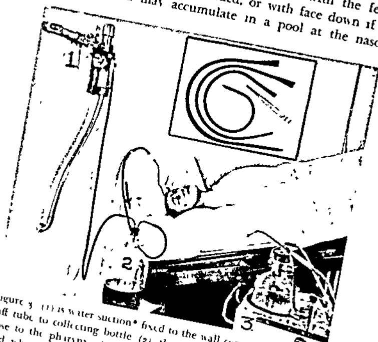


Figure 3 (1) is water suction* fixed to the wall connected by a fairly stiff tube to collecting bottle (2), this to catheter through patient's nose to the pharynx (3) is a standby mechanical suction Beveled and whistle tip catheters are shown in insert (Reproduced with permission from the *Laryngoscope* June 1951)

pharynx hypopharynx or cheek There it may be picked up as quickly as it covers the opening of a perforated rubber tube connected to continuous water suction Remembering the few minutes of anoxia necessary to produce irreversible damage it must be obvious that

* Manufactured by the S M R Co Los Angeles Calif

dependence cannot be placed on intermittently applied mechanical suction. We know of no mechanical apparatus which will bear constant use except one attached to a vacuum tank or with continuous water suction The device pictured (see Fig 3) is easily attached, effective in the pressure desired, almost foolproof and does not get out of order. Some plumbing codes interdict its use, yet if it is connected to a half liter collecting bottle filled with antiseptic solution, contamination of the general water system seems impossible.

Thirty years of experience with postural drainage in respiratory obstruction, especially in acute infectious tracheobronchitis, has persuaded me that it is of the utmost value and without danger if used early Lateral x-ray with radiopaque oil outlining the tracheobronchial tree indicates that elevation of the foot of the bed in the supine position must be at least 17 to 20 degrees to prevent flow of secretion into dependent lung bed (Fig 4). We have advocated 35 degree elevation but now believe that is extreme and seldom obtainable. Twenty to twenty-three degrees is usually satisfactory If the patient will lie prone with the head to the side, as usually only infants will do, drainage is effective and normal physiology is not disturbed. By the use of special foam rubber supports the face down position has been made easier to maintain (83) If this can be made comfortable so that the patient will easily accept it, it should be valuable. We have found it difficult, however, to get adult patients to cooperate with this position It is, of course, important also to shift the patient's position frequently in order to prevent congestion and atelectasis in dependent portions of the lung

Bower (76) and his group have raised a question as to

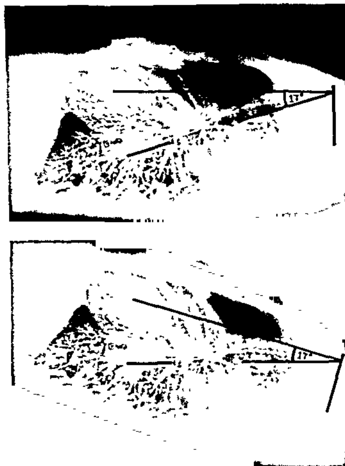


Figure 4 The trachea and bronchi are outlined with radiopaque oil A represents the declivity of trachea and main bronchi from body axis B shows minimum elevation desirable (Reproduced, with permission, from *Arch Otolaryng*, *46* Aug 1947)

the safety of the Trendelenberg position in seriously ill patients. They found in a limited number of patients that at an elevation of 15 degrees for 15 minutes the average drop of tidal air was 20 per cent. This, however, left an average tidal air of about 350 ml. on which a patient at rest, I believe, may be carried, especially if aeration of the lung is uniform and if adequate oxygen is given. My experience with postural drainage for over 30 years in the treatment of acute laryngotracheobronchitis has been highly favorable. The dangers do not seem to outweigh its great benefits in a patient who may have important amounts of secretion or fluid in the trachea or bronchi. It helps to insure that the lower airway is not flooded and that the whole alveolar area is accessible to inspired air; that the absorption of oxygen and excretion of carbon dioxide is well distributed throughout functioning lung; that areas of atelectasis with or without pneumonitis do not develop. It is true that long continued postural drainage must be used with caution in patients with marked vasomotor disturbance or possible cerebral edema. To judge by the patient's response the procedure is usually beneficial, often dramatically favorable

NUTRITION AND FLUIDS

Fluids and nutrition are to be maintained parenterally while there is any danger of flooding the airway because of disturbed deglutition Sufficient fluid must be provided to carry on normal metabolism and to prevent dehydration Secretions should be kept normally thin so they may be more easily removed from the respiratory tract, especially if tracheotomy is done. Excess fluid may overburden the circulatory system and promote cerebral or pulmonary edema Due allowance must be made, however,

for the 1500 to 2000 cc. or more of fluid usually lost in saliva, inflammatory secretion or by regurgitation in the patient who is unable to swallow. Adults require about 3000 to 3500 cc per day.

ELECTROLYTE BALANCE

It is especially important that electrolyte balance be maintained This can be discussed only briefly. Some aspects of the question are also too incompletely understood or too controversial to be covered here Sodium chloride is necessary but because when given alone it tends to increase the loss of potassium, Gamble (84) suggests that the customary ratio of normal saline or Ringer's solution might well be further reduced We have usually given fluid in the ratio of one part or less of saline to three parts of water or glucose solution One patient had marked edema of the face and neck and probably of the brain as judged by depression of the sensorium This had been ascribed to postural drainage but cleared up rapidly on withholding sodium

The importance of proper levels of potassium in the cell—and it is concerned chiefly with intracellular metabolism—has only recently been investigated and appreciated Low concentration leads to cardiac and general muscle weakness High concentrations may be lethal and cause cardiac death (85) The range of the critical level may be very narrow Deficiency in potassium was found usually in poliomyelitis by the Los Angeles group This can arise from cell destruction, from loss through saliva and other secretions and probably in acidosis But in poliomyelitis they demonstrated increased retention with renal impairment Levels rose from the normal 16 to 22 mgm % to between 28 to 50 mgm %. Both anoxia and alkalosis due to hyperventilation may lead to renal im-

pairment and nitrogen retention. Adrenal damage by anoxia might lead to disturbed potassium balance since hormones of the type of desoxycorticosterone are potent in causing potassium excretion, and adrenal cortical insufficiency leads to a decrease in potassium excretion.

Darrow (86) has stated that his solution containing potassium may be safely given subcutaneously and has reported favorable results with it Bower (87) has given potassium intravenously, but has carefully regulated its use by blood determinations.

Hoyne and his associates (88) reported dramatic results in weak and lethargic patients to whom potassium was given intravenously. It was administered in dilutions of two to three grams per liter and given slowly at the rate of 8 to 12 cc. per minute Potassium serum levels were very carefully controlled by the use of the photometer. When levels became normal parenteral potassium chloride was reduced to 4 grams per day. This was stopped as soon as the patient could be fed

Fox (89) and his group have recently proposed a physiological replacement solution which contains sodium, calcium, magnesium and chloride ions in normal plasma concentrations Its potassium concentration is twice normal value and bicarbonate is provided in double its normal concentration in plasma Their data indicate that this solution prevents hypopotassemia without danger of toxicity, corrects moderate acidosis without inducing alkalosis, replaces extra-cellular electrolytes and induces copious secretion of urine and salt. Its use in poliomyelitis seems reasonable

Gamble (84) says, however, that "the danger of damage to the heart is so serious that I should not like to see the parenteral use of potassium go at once into clinical currency " If so it should be controlled by careful routine

analyses best done by photometry which appears to be reasonably accurate

If, however, potassium can be given by gavage or swallowed the organism appears to be able to absorb and retain the proper and safe concentration of this electrolyte from the gastrointestinal tract That is a good argument for earlier use of tube feeding, possibly by the third day, if a tracheotomy has already been done to lessen the danger from regurgitation The feeding tube also permits aspiration in the occasional gastric hemorrhage. On account of the danger from vomiting, we still choose to use only parenteral administration of fluids, glucose, electrolytes, vitamin C and B complex and protein hydrolysate until patients can swallow.

VITAMINS AND PROTEIN

For repair of tissue damage and the tracheotomy wound, and to avoid edema and promote general well being, it is essential to prevent vitamin deficiency and hypoproteinemia

Vitamin C is given from the outset, but we have not thought there was special benefit from very large doses. Vitamin B complex has been given parenterally after the first week Some experimental work in mice (90) has indicated that the virus of poliomyelitis was less virulent when vitamin B was deficient This was held to be an indication that the virus thrives best in healthy host cells That might explain some recent cases where vigorous healthy young men proved to be our poorest risks

Bower and his associates (87) demonstrated that serum albumin levels drop early in poliomyelitis between the first and third day and progress unfavorably for at least ten days They found also that the serum globulin ratio was likely to be reversed They believe this drop in serum

albumin is an important factor in the development of edema, especially of the brain. This edema, they postulate, tends to further diminish circulation to brain tissue and thus increases anoxic effects They found this drop in serum albumin retarded or prevented by the use of pooled irradiated blood plasma with apparently considerable benefit to the patients. We have used plasma with benefit in severe cases especially where the patient's condition approached that of shock We give a protein hydrolysate or amino acids by vein after the second or third day in addition to glucose from the outset. Gavage is begun after some return of ability to swallow starting with 50 to 75 cc. increased to 300 to 400 cc. per feeding alternating with water and formula every hour. A balanced formula is used which gives a minimum maintenance requirement. In the adult this is at least 1800 calories daily.

TRACHEOTOMY

While the measures already outlined will carry many patients to recovery, in others certain danger signs point to the need for further relief Tracheotomy was apparently first suggested by Wilson (61) for use in some circumstances in bulbar poliomyelitis, but only in recent years has it come to be widely employed. A clear concept of conditions which make it necessary, more precise indications for its employment, the almost complete lack of danger in its use and the fact that it is life saving in a large percentage of cases have come to be generally appreciated.

Probably its greatest value comes from the fact that it bypasses secretion in the pharynx and provides a clear airway (see Fig 2). Although Wilson (91) has been conservative in his advocacy of tracheotomy he has well stated its value. *He said, "Tracheotomy is a radical step but often* lifesaving Its purpose is to furnish a free airway so that the air necessary for respiration does not need to be moved

down a pharynx full of secretion and through a larynx continually in spasm." I believe the observation quite correct although I cannot agree with his condemnation of this simple operation by calling it radical under such circumstances. Secretions in the trachea and bronchi may easily be removed by suction through the tracheotomy tube, especially if the patient is placed in the position of postural drainage After tracheotomy the use of the respirator is safe and this may be important in a depressed patient

Greater use of tracheotomy was advocated in 1943 (62) and an attempt was made to rationalize the procedure. Since then reports of large numbers of operations by Priest, Boies and Goltz (65), by Bower (76) and others offer definite proof that it is of great value and a relatively harmless procedure

INDICATIONS FOR TRACHEOTOMY

Our indications for tracheotomy in poliomyelitis are:

1. Progressive anoxia in spite of other treatment

2 Coma, irrationality or marked restlessness or other signs of severe anoxia, not immediately relieved on suctioning

3. Marked restlessness or stupor in a patient in a respirator, even if the disease appears to be spinal.

4 Fluid accumulation not certainly taken care of in any patient.

5 Marked spasm (or bilateral paralysis) of vocal cords.

6 Rapidly progressive bulbar symptoms or vasomotor paralysis

7 Bulbar involvement in any patient requiring a respirator

8 Low tidal air not corrected by aspiration or safe respirator pressures

9 Untrained attendants, inadequate equipment or poor

cooperation with any doubt that airway will be kept free of secretions.

These indications merit further discussion.

Symptoms of anoxia must be closely watched for, including restlessness, dyspnea, disorientation, mental depression, increased blood pressure, or later fall, and cyanosis. No patient should become unconscious under observation.

Under the third indication it is not always possible to diagnose exactly the type of involvement, especially because an involvement originally spinal may spread quickly to the bulb. Moreover, fluid may accumulate not only from pharyngeal paralysis but because of bronchial or pulmonary inflammation or pulmonary edema.

Additional indications have been given Sudden rise in blood pressure has been considered as evidence of carbon dioxide accumulation It has been observed that if blood pressure readings taken at half hour intervals showed a rise of 15 to 20 points, tracheotomy gave early relief from developing anoxia On the basis of physiologic factors I am willing to believe that this might be an important indication

We are not quite ready to recommend routine prophylactic tracheotomy if the clinician is prepared to intervene at any moment and realizes how rapidly such patients can fail But it is much better to do tracheotomy unnecessarily in many cases rather than too late in a single one, and too early rather than after irreversible damage We have seen no important complication from the operation, nor any case in which it contributed to an unfavorable outcome That should be universally true if proper technique is used This is discussed in a following chapter The high operation should be done—usually *through the*

first and second interspace and the second or third trach ring to permit later use of the respirator if that becoı necessary.

Tracheotomy is not to be withheld, as it is too often, the last forlorn hope. It should be done more often wit physician's first experience with the disease, as mı greater skill, training and equipment are required to ca on without it As Strobel and Canfield (82) state, ever very ill patients delay of tracheotomy is more danger than its performance. They point out also that trach omy permits a patient in a respirator to swallow m easily because the usual pause in respiratory action (ing which a patient swallows is not possible with n tank respirators.

Postoperative care of tracheotomized patients is e cially important since cough is impaired or absent, e cially if they are in a respirator For such a patier trained resident or interne should be on the floor at times Great gentleness to avoid trauma is required. W continuous water suction from the pharynx is contin through the nose, intermittent suction is used through tracheotomy tube, removing the inner cannula if ne sary A soft whistle tip catheter is gently used or a rubber catheter obliquely cut and smoothed in a flame with a small second opening near the end to bı excessive grasp of mucosa, while permitting remova secretion or plugs Dew point humidification, gentle rigation as indicated with warm three per cent soda bi bonate solution followed by normal saline, both remc by suction in postural drainage will usually pre atelectasis and obviate the need of bronchoscopy

Wetting agents (sodium lauryl sulfate) have been u but I have seen one case in which they caused foan

which contributed, I believe, to an unfavorable outcome. Grace (92) has added wetting agents directly to the water in the mechanical humidifier with what he thinks is a better effect. Forceps technique is employed for asepsis Antibiotics are used but not long enough to permit the overgrowth of monilia and other fungi. If penicillin and streptomycin are used instead of the broad spectrum antibiotics this danger appears to be greatly lessened. Sulfonamides are, we believe, sometimes harmful in poliomyelitis

I have not taken a definite stand for prophylactic tracheotomy believing that with experience we could anticipate the need for intervention in time It had even seemed that with an appreciation of the mechanics involved and with meticulous care a patient with some difficulty might be safely carried in a respirator with postural drainage and continuous aspiration. Strobel and Canfield's (82) advice to operate when there are early and progressive bulbar symptoms, or when a long siege in a respirator is likely with any fluid accumulation now seems to be sound

A growing belief in the importance of laryngeal spasm even with small amounts of fluid in the pharynx, unhappy experience with reluctant consultants and untrained and skeptical house staff on whom final dependence rests for proper treatment, and delayed requests for help make me now think it should be a fixed rule that any patient going into a respirator with any difficulty with secretion should have a tracheotomy.

On studying our own records I was shocked to find that of nine patients with bulbar symptoms who were placed in a respirator without tracheotomy at any time, only one survived Jacobson, Cohen and Carter (93) studied roentgenologically pulmonary complications in relation to tracheotomy in respirator patients. Of 26 patients who had

tracheotomy the same day they were placed in the respirator only 14 developed atelectasis or other demonstrable changes. Of 17 who went into the respirator one day before the operation was done, 14 had such complications. It is not easy to detect beginning atelectasis even with x-ray (22) nor always practicable to use it When once such a pathologic condition in the lung is established, it is also difficult or frequently impossible to correct it. The conscientious physician cannot easily brush off the self reproach that will follow the postmortem finding of extensive atelectasis that developed because he has done nothing because symptoms, though definite, were not animate enough to slap him awake It is certainly much better to perform such an easy and safe procedure as tracheotomy many times too early or unnecessarily than to have failed to do it once in time to prevent serious consequences

THE RESPIRATOR IN BULBAR POLIOMYELITIS

It was at one time considered useless and dangerous to use the respirator in bulbar poliomyelitis, but now it seems to be widely employed in that disease This employment has closely coincided with more widespread adoption of tracheotomy in treatment We have found in bulbar poliomyelitis that it may be of tremendous value and without danger if a clear airway is insured It is easy to see that if the upper airway is filled with viscid secretion and suction produced below it by a normal or mechanical respiratory mechanism, the air column will tend to push ahead of it a diaphragm of viscid secretion Some air will bubble through This action is shown by the model illustrated in Figure 2 It is evident that such a procedure might easily drown a patient in his own secretions It will be indicated later in this chapter how such negative pres

sure against obstruction also tends to produce pulmonary edema.

It should be equally clear that if such secretion is by-passed by a tracheotomy so that changes in pressure are equalized, or nearly so, within and without the area of ventilation no fluid will be drawn in The immediate relief of depressed patients and their usual adaptation to the rhythm of the machine, as well as the greatful rest which they then enjoy, offer excellent proof of the respirator's safety and value.

Bulbar patients may need a respirator for several reasons. Occasionally we have seen a primarily spinal type of paralysis spread up to involve the medullary center. During the last year the process has seemed more widespread and purely classifiable types less frequent. Cases seen in 1951 have shown some bulbar involvement and some weakness of the respiratory muscle control at the same time. Our experience has indicated, however, that most often the medullary center is depressed by anoxia and carbon dioxide, and that the depression may have become rather profound before the anoxic factor is recognized and corrected There is usually a serious degree of exhaustion before action is taken. This is due in part to the muscular effort in breathing against obstruction In addition there is on the nervous and mental side considerable tiring and depression by the conflict between reflex and voluntary mechanisms which strive at the same time to ventilate the lungs yet prevent the aspiration of secretion. Also the respirator may, if the medullary center is out, tide over the patient during the difficult switchback from control of respiration through the chemoreceptors, effective under hypoxia, to a recovered medullary center which will normally respond to carbon dioxide and changes in hydrogen ion concentration Again the fact,

that usually in the serious cases, relief was rather rapid on clearing the airway and instituting use of the respirator, indicates that anoxia and not virus infection of centers was the vital factor. It is repeated that it should be the rule that tracheotomy should be done on the patient with accumulating secretions before he is placed in the respirator

Wilson (94) has well described the general management of the respirator, and we have rather closely followed his directions We, however, feel that the respirator is both safe and very valuable in bulbar poliomyelitis We follow Wilson's advice to initiate rates of 15 to 20 per minute for adults and 20 to 30 for children, but we allow the patient's sense of well being to dictate moderate changes The slightly faster rate seems to be better accepted in the beginning, but we have thought that a slightly lower rate is more physiological and permits more complete diffusion of air in the lungs and greater change in the dead space We have used negative pressures as a rule between 15 to 18 and occasionally to 20 cm of water The positive pressure adjustment of the tank respirator seems of little or no value That, however, is not true of intermittent positive pressure such as it provided by the Bennet apparatus and synchronized with the negative pull of the tank This may be of considerable help in desperately ill patients, especially when pulmonary edema is threatened

In unobstructed patients rates and settings described seem to give adequate ventilation to judge by the patient's comfort, absence of symptoms of anoxia, and good volume of tidal air as measured by the ventilation meter Except for short intervals greater negative pressure may slightly increase the danger of pulmonary edema, interstitial emphysema or pneumothorax.

Great caution should be used in attempting to secure

relief of discomfort, distress or symptoms of anoxia by marked increase in the rate or depth of respiration. Failure to secure adequate ventilation at or under 20 cm. of water of negative pressure should raise a question of blocked airway, atelectasis or beginning pulmonary edema. Every other means including aspiration and probably positive pressure should be used to restore proper ventilation without unduly increasing the setting of the machine As described later higher pressures quickly released may help relieve atelectasis.

OUTLINE OF TREATMENT

Treatment at the Evanston Hospital is outlined·

1. Postural drainage with continuous water or vacuum tank suction instituted at beginning of swallowing difficulty.

2 Parenteral fluids, electrolytes, vitamin C and glucose followed later with intravenous vitamin B complex and protein hydrolysate or amino acids

3 Oxygen administration, but as an adjunct to clearing the airway and eliminating carbon dioxide

4. Tracheotomy when signs of anoxia persist or develop in spite of the above

5 The respirator when respiration is depressed from anoxia, exhaustion or any other cause.

6 Antibiotics against infection (but not to the point of overgrowth or fungi)

7 Careful nursing and great care to maintain morale, to allay fear and to keep the patient reassured.

Such treatment should be usually effective if undertaken early Certainly even without much experience with the disease these measures are to be preferred to special

apparatus which perhaps places wrong emphasis and delays proper measures. These need special discussion as some are very valuable, especially in serious cases

Oxygen has proven of great value in respiratory obstruction It should, however, be emphasized that much more important are the measures taken to provide for the elimination of carbon dioxide. A clear airway is the prime need and no special device to provide oxygen should divert attention from this

SPECIAL APPARATUS

The oximeter has been proposed to determine oxygen need It needs to be set against a normal base, is said by some not to be a reliable apparatus, gives no direct indication of carbon dioxide excess, and in our experience is chiefly of value in determining when a chronic respirator patient may leave the respirator

The electrophrenic respirator has some value in emergencies but does not have the applicability or dependability of the tank respirator It is not well tolerated for long periods and does not seem to warrant the emphasis given it Bower (95) watched a trial of this device and discarded it as impractical

Various portable respirators are of definite use in doing tracheotomy and in weaning the patient from the tank respirator They do not yet have the dependability or efficiency of the tank respirator

The rocking bed seems to have proven of real value in making the convalescent patient independent of the respirator and shortening the transition period weeks or more It can not replace the tank respirator in a patient with serious respiratory depression

Attempts at determination of chemical acidosis in our experience have not replaced careful clinical observation. Respiratory acidosis, as pointed out by Gamble (96), gives opposite blood findings from those of metabolic acidosis and must be interpreted in the light of blood pH and compensatory mechanisms. To be significant, tests must be done on alveolar air, here impossible, or on arterial or capillary blood. We have not found them of great practical value in individual treatment, but they are very important for research and to study the efficacy of methods.

The Los Angeles group, under the lead of Bower and Bennet (76), have designed a number of mechanical devices which careful physiologic studies indicate have much promise. They are only now becoming generally available. They include:

1. The Bennet ventilation meter which aids in determining the setting of the respirator. It also shows when dangerous lessening of the tidal air indicates need of additional help by tracheotomy or the respirator. We have found this easy to use and of considerable value.

2. The intermittent positive pressure attachment which synchronizes through mask or tracheotomy with the negative pull of the respirator Some physiologists insist that in any case the true value of the negative pressure is the same as the algebraic sum of combined positive and negative pressure However, the Los Angeles group seem to have demonstrated that the combined positive and negative pressure is definitely beneficial in more wide inflation of lungs and prevention or relief of pulmonary edema and atelectasis They found that when using the two together usually a smaller total pressure is required which is better tolerated by the patient Motley (97) believes that the reason oxygenation of the blood may be improved when

intermittent positive pressure is used to supplement the tank respirator is that there is a more uniform aeration of partially atelectatic areas.

3 The flow sensitive breathing units of Bennet appear to be valuable for emergency use, especially for tracheotomy, which can then be done out of the respirator. It also provides useful positive pressure for use in pulmonary edema.

4 The cam adjustment appears to make a more comfortable and normal timing of the respiratory cycle.

The question of transportation of bulbar patients requires special consideration If patients are near a poliomyelitis center they can be better treated in hospitals properly equipped, by teams properly trained, since some patients may first be lost while experience is being gained by a group to whom the problem is new. But travel and strain are so injurious to these patients except at the beginning of bulbar symptoms that they might do better to be treated locally, even by men of less experience, than to undergo time consuming and exhausting travel

While early pregnancy may add little hazard, poliomyelitis in advanced pregnancy is a special problem with a rather unfavorable experience until lately. The gravid uterus near term distends the abdomen, interferes with respiratory movements, embarrasses the heart and makes postural drainage poorly tolerated If early preliminary tracheotomy is done, and the uterus emptied by Caesarean section, these patients seem to respond quite well to the treatment outlined

OTHER COMPLICATIONS

Emphasis has been put on the early and decisive relief of obstruction to prevent complications because if once

established it may be extremely difficult or impossible to correct. Especially is this so of atelectasis and its train of impaired respiratory function and secondary infection Likewise, pulmonary edema when once initiated in poliomyelitis may be irreversible.

ATELECTASIS

Some factors in the development of atelectasis have been discussed in Chapter II. Obstruction by spasm and secretion and impaired circulation operate especially adversely in poliomyelitis, and most patients dying of this disease present findings of atelectasis The condition may be hard to diagnose, but it is to be suspected when a patient previously doing well develops dyspnea and other signs of anoxia If these persist after the airway is cleared, and the ventilation meter then shows diminished tidal volume, the presence of atelectasis is more likely. If any important area of the lungs is not aerated we have found, especially in tracheobronchitis, that with the stethoscope over that area the breath sounds are muffled or absent. Roentgenograms may be of limited value since they will be taken under trying circumstances with a portable machine. Anspach's (22) work indicated that unfortunately atelectatic areas usually did not give a shadow until infection was already present.

To prevent atelectasis the airway must be kept clear, if necessary by tracheotomy, and the pulmonary circulation preserved by adequate lung movement. Frequent change of position helps prevent stagnation and congestion of blood in dependent portions of the lung.

Galbraith's and Steinberg's (14) experiments on dogs cited earlier, indicated that atelectasis after 24 to 48 hours might not be cleared even by relief of obstruction, espe-

cially if secondary infection had caused pneumonitis In the human patient with good nursing care and the use of antibiotics there is probably somewhat more leeway. Each hour the condition persists, however, adds to the difficulty of its relief and to the danger from anoxia and exhaustion The first indication is to clear the airway and to remove the obstructing secretion or mucus plugs, either by aspiration through tracheal catheter or by bronchoscopy But usually, if atelectasis does develop, this means that tracheotomy should probably have been done earlier and should not be further postponed

After tracheotomy suction by catheter is frequently successful, especially if saline or dilute soda solution or penicillin is used to thin, soften and dislodge viscid secretion If this is not effective bronchoscopy with a small tube may be done without hesitation after the tracheotomy tube has been removed This manipulation should be done so gently that the mucosa is not traumatized and should not be repeated unnecessarily

In an attempt to simulate cough and augment any tussive power remaining Strobel and Canfield (82) used pressure on the thoracic cage in the stage of expiration by the hands inserted through the tank ports

An effective simulation of coughing with dilatation of bronchi and expulsion of secretion has been reported by Barach and his associates (98) A flow rate of high velocity was obtained by first building up a peak negative intratank pressure of 40 mm of Hg and then abruptly terminating it by instantaneous opening of a special valve For this a device called the "exsufflator" is used in connection with the tank respirator Colleagues who have seen this mechanism used are enthusiastic about the dramatic relief they have seen with it

PULMONARY EDEMA IN POLIOMYELITIS

Pulmonary edema in poliomyelitis has been sometimes ascribed, along with vasomotor and cardiac disturbance, to direct action of the virus on brain stem and autonomic ganglia. Although the neurogenic theory has been used to explain the pathogenesis of pulmonary edema in skull injury and subarachnoid hemorrhage, others have considered the effects due to secretional obstruction and laryngeal paralysis or spasm. The controversy is not yet settled.

But for poliomyelitis Polley (99) has assembled data, for which he provides good documentation, to explain pulmonary edema without directly involving factors of brain infection or damage. It can be explained on the mechanical effects of hydrostatic and osmotic pressure in the pulmonary circulation, plus greater negative intrapulmonic pressure associated with obstruction. To this may be added increased capillary permeability due to anoxia and carbon dioxide excess as shown by Drinker. Associated cardiac failure may further predispose to edema, as may also serum albumin deficiency

The function of systemic capillaries is to surrender nutrition, electrolytes and fluid to general tissue cells and take up metabolites Excess fluid is removed by lymph flow On the other hand in the lungs respiratory gas exchange is seriously hampered by fluids. Here excess cannot easily be disposed of by its relatively scanty lymph flow Especially is this so when there is limitation of active lung movements on which blood flow depends.

A chart modified from Polley indicates varying conditions of hydrostatic and oncotic pressure in systemic and pulmonary capillaries This is described in the text which follows

HYDROSTATIC AND ONCOTIC (OSMOTIC) PRESSURES OF CAPILLARY CIRCULATION
(After Polley)

	Filtration Pressure	Oncotic (Retaining) Pressure	Result
A Normal Systemic	22 mm Hg hydrostatic pressure	15 mm Hg oncotic pressure	7 mm Hg filtration driving force
B Normal Pulmonary	10 mm Hg hydrostatic (negligible intrapulmonary pressure)	25 mm Hg oncotic pressure	No filtration 15 mm Hg opposing filtration
C Pulmonary Circulation with Closed Airway	10 mm Hg hydrostatic 40 mm Hg negative intra pulmonic 50 mm Hg filtration pressure	25 mm Hg oncotic pressure	25 mm Hg filtration driving force
D Pulmonary Circulation with Respirator—Airway Occluded	10 mm Hg hydrostatic 20 mm Hg effective neg intrapulmonic pressure	25 mm Hg oncotic pressure	5 mm Hg filtration driving force

As outlined in the table from Polley (A) the systemic capillaries have a relatively high hydrostatic pressure of about 22 mm Hg They are surrounded by tissue cells and interstitial fluid so that the oncotic or osmotic balance tending to retain fluid is approximately 15 mm. Hg This leaves the filtration force of 7 mm Hg driving fluid out of the capillary.

On the other hand normally in the greatly widened lung bed the hydrostatic pressure is held to be as low as 10 mm Hg. (B). The alveolar cell lies against capillary endothelium without intervening interstitial fluid and oncotic pressure is about 25 mm. Hg. With unobstructed airway intrapulmonic pressure approaches zero. The difference, 25 minus 10, leaves a force of 15 mm. Hg. opposing filtration. This secures the fluid free surface at which gaseous interchange may take place. This force is so effective under normal conditions that Colin in 1873 was able to give a horse 21 liters of water intratracheally in three and one-half hours with no ill effects

With obstruction of the airway the effect is quite different (C). The negative intrapulmonary pressure in forced inspiration may go to −40 mm. Hg. or higher. This would leave a filtration pressure of 25 mm. Hg. tending to force fluid into the lungs While such pressure might cause little harm in a short time in normal lung with good lymph flow, it seems reasonable that it might, if continued, be disastrous if the cells are injured by anoxia, hypercapnia and impaired circulation. If such obstruction persists pulmonary edema would be expected to occur.

The filtration driving force in a respirator set at −20 is 5 mm. Hg which is not great (D), but if multiplied by the approximately 1000 cycles per hour of the apparatus it could be important It certainly allows no safe margin if any obstruction is allowed to occur. It is also a warning against an attempt to compensate for poor ventilation by undue increase of the negative tank pressure.

The conditions with a smoothly working respirator, tracheotomy and free airway are the same as shown in condition B, that is, approximately normal

Pulmonary edema is a relatively common finding post mortem. It is almost irreversible although Kernan and Barach (13) and Bower, Dillon and Bennet (76) have

shown that it may be favorably influenced by positive pressure.

Recently the successful use of anti-foaming agents has been reported in pulmonary edema. For this purpose Gootnick, Lipson and Turbin (100) found ethyl alcohol inhalation effective when used by mask in a vaporizer through which oxygen is passed.

Conversely, it would seem advisable that foaming agents such as sodium lauryl sulfate (Duponol) should not be used to aid removal of secretion where there is any danger of pulmonary edema. I saw increased foaming and a bad result in one case in which it was used.

Again, as with other complications in poliomyelitis, pulmonary edema is much easier to prevent than it is to control when once it is established. Its occurrence signals the failure of compensatory and defensive mechanisms and all too often means defeat. The mechanical effects of obstruction and the chemical effects of anoxia and hypercapnia should be foreseen and forestalled long before they eventuate in pulmonary edema.

CONCLUSIONS

We conclude that the serious effects in bulbar poliomyelitis are not primarily due to overwhelming infection of the vital centers in most cases.

If it is looked upon as a problem in secretional obstruction with resulting anoxia and carbon dioxide accumulation, this disease lends itself to treatment that should be favorable in an increasing percentage of cases.

This is a problem for a team of which one member should be fully trained in the problems of respiratory obstruction including bronchoscopy if possible. He should be allowed to see bulbar patients at the first sign of swallowing difficulty and share fully in any decision made for or against further intervention.

CHAPTER V

TRACHEOTOMY – ITS EXPANDED ROLE AND TECHNIQUE

UNTIL RECENTLY tracheotomy had been held in reserve as a rather hazardous and reportedly radical procedure to be used in bulbar poliomyelitis and other conditions rather as a last resort. However, in several papers reasons were given for considering it a relatively innocuous and effective procedure to use in respiratory obstruction before irreversible pathologic changes developed Long before this many physicians working with tracheobronchitis and other obstructive conditions had realized the relative simplicity and frequently life-saving effect of the operation In recent years it has come to be accepted by men in many fields, including internal medicine and contagious disease, anesthesia, surgery and neurology as having a wide field of application

The good results from tracheotomy may be summed up as follows

1 It prevents immediate asphyxia from obstruction, spasm, paralysis or flooding secretion

2 It prevents anoxia and carbon dioxide accumulation with brain damage and general and medullary depression.

3. It prevents exhaustion by reducing respiratory effort.

4. It permits removal by suction of secretion, plugs and crusts If necessary, they may be removed by irrigation from trachea and bronchi.

5 It bypasses secretion and permits deglutition to function during respiration

6. It greatly limits changes secondary to obstruction such as atelectasis, pneumonitis and pulmonary edema.

7. It permits the use of the respirator in the presence of accumulation of secretion and/or spasm.

The danger from the operation itself, which too often has not been separated from that of the disease requiring it, has been shown by many men including Jackson (101), Negus (102), Figi (103), Von Leden (104) and others to be very small, having a mortality of only a fraction of 1%. If done before the patient's condition is critical it is usually rather easy and any graduate in medicine should be able to accomplish it in an emergency. The chief difficulties are associated with hemorrhage, infection and emphysema. It may be done deliberately if first a bronchoscope or endotracheal tube is passed by one skilled in its use to aspirate secretion and to relieve asphyxia so the patient is relaxed and not struggling. If these helps are not available delay is much more likely to be serious.

Some points of technique will be presented. Local anesthesia is used employing 0.5 to 1.0% of procaine solution with two to five drops of 1:1000 adrenalin chloride per ounce. This reduces hemorrhage and helps to provide a dry field. A lozenge shaped area of approach is outlined by injection with a fine needle and then the line of incision infiltrated, including the pretracheal fascia. Injection during withdrawal of the needle or frequent aspiration are done to insure against injection into blood vessels.

General anesthesia is extremely dangerous in conditions requiring this intervention. It may add further serious depression, especially to the medullary centers. It takes more vital time. It obtunds the cough reflex and destroys the patient's power to expel secretion. In comatose or seriously depressed patients relatively small

amounts of local anesthesia are required. If young patients struggle or are antagonistic it is usually because of anoxia. This can be prevented by preliminary bronchoscopy, or such patients can be controlled by mummying.

The shoulders should be raised on a flat pad, and the head extended well if that does not increase the difficulty in breathing. It is important that rotation of the head and neck be prevented so that exact midline orientation is maintained. It is well to remember Jackson's tracheotomy triangle lying below the level of the cricoid cartilage and within the median borders of the sternomastoid muscles with its apex at the suprasternal notch. No vital structure should be injured in that area, and the chief difficulty would be bleeding from the thyroid isthmus. The incision should be in the midline beginning below the thyroid notch which may be palpated by the forefinger with the thumb and middle finger fixing the thyroid cartilage for orientation. The emergency seldom justifies the collar incision except in some neck injuries. The primary incision should extend only far enough to give adequate exposure without an undermined wound in which emphysema and infection are more likely to occur

Except for one horizontal incision through the thyroid fascia to mobilize the isthmus, all incisions should be made in the midline The strap muscles are separated exactly in the midline and the pretracheal fascia incised in that line so that no cartilage is left denuded. To do so increases the risk of perichondritis The isthmus may be pushed up or down although I prefer to lift it on curved Kelly forceps, cut vertically through it with scissors and transfix the ends with suture ligatures. Hemorrhage into the trachea is an important complication to be avoided.

If the tracheotomy tube is to be worn a long time, the trachea should be opened in the third or fourth ring. If there is a possibility of a respirator being needed, the

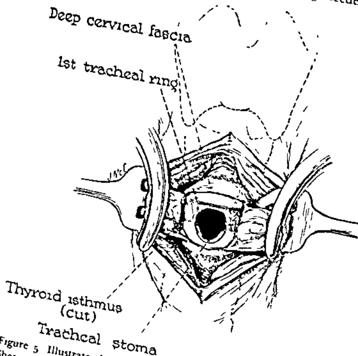


Figure 5 Illustrates high tracheotomy to permit use of respirator Shows exact midline incision with no undermining or denudation of cartilage Cut ends of isthmus are caught in forceps for suture ligature Ovoid of cartilage excised to provide watertight fit of tube

stoma should be at the level of the first interspace and second ring or one ring lower, so that the tube may lie well above the collar of the respirator for easy aspiration

and care. I prefer to make a small transverse cut in the interspace and remove an ovoid of cartilage through which a tight-fitting tube will just pass with a nearly water-tight connection (Fig. 5). Jackson's cartilage hook retractors are useful for this maneuver as well as for lifting and holding the trachea in the wound while it is opened. If time permits, the needle of a hypodermic springe is passed into the trachea to identify it by aspiration of air. Preferably a few drops of 2% to 8% cocaine, depending on the patient's age, are then injected into the trachea to allow tranquil acceptance of the tube.

The tube should be long enough and of a suitable curve so that it rests smoothly in front of or against the posterior wall without pressure or irritation. At first, the largest tube easily taken permits easier breathing, aspiration and care Later as the airway becomes more free a smaller tube shunts more air through the larynx and causes less drying and allows more effective removal of secretion by coughing.

Packing is placed rather tightly below the tube to prevent bleeding, emphysema and infection, and it is usually removed after two days A smaller pack is put above the tube for hemostasis and removed after one day. For packing I prefer a cod liver oil preparation such as "Gadamint" on strip gauze as it seems to cause less irritation and infection I formerly taught that leaving the wound open was less likely to cause emphysema and infection, but now close rather tightly around the tube to hold the packing tightly in place. Less scarring results and apparently less emphysema and infection.

Emphysema, it has been rather well shown, can usually be prevented if, by passage of bronchoscope or endotracheal or Mosher tube, an open airway is guaranteed so that during operation air is not drawn into tissue spaces

by increased negative thoracic pressure Rather to my surprise no important emphysema has occurred in our poliomyelitis cases even when the respirator has been used It must be remembered that emphysema of the neck and mediastinum may be associated with atelectasis, with secondary interstitial emphysema or be due to rupture of blebs at the hilus or under the pleura Holt (29) years ago reported an influenza epidemic in which emphysema and pulmonary collapse were common complications without any intervention Those opposing tracheotomy are hardly justified in citing the dangers of emphysema, collapse or injury to the pleural dome as due to the operation

Bleeding is to be avoided, if possible, by careful hemostasis which also permits easy orientation and dissection When time permits the field should be quite dry before the trachea is opened, although if the situation is urgent, packing will usually control bleeding

In spite of all forethought and care, emergency stab tracheotomy is occasionally required To perform it, the patient is held with the chin exactly in the midline, the neck extended, but *not* to the degree that the trachea is collapsed so the esophagus might be injured The thyroid notch is identified if possible, and the thyroid cartilage fixed between thumb and middle finger A sharp blade carries a moderate length incision down to and through the trachea preferably high, but below the cricoid which never should be injured as it is the one continuous ring preventing collapse of the larynx Air will bubble in and out identifying the trachea and giving relief from asphyxia Excessive bleeding must be controlled by the non-operating forefinger while a tracheotomy or improvised tube is placed with packing around it.

CHAPTER VI

TRACHEOTOMY IN OTHER DISEASES WITH RESPIRATORY OBSTRUCTION

IT HAS RECENTLY come to be recognized that respiratory obstruction plays an important role in many conditions where anoxia and changes secondary to obstruction add a critical overload to an already depressed organism If these factors are recognized, patients previously condemned as hopeless may be saved by treatment directed to the relief of obstruction. Proper posture and change of position, temporary airway, oxygen and catheter or bronchoscopic aspiration may be enough in many cases if patients are not overwhelmed by the essential disease. If these measures are not sufficient tracheotomy may be life saving

Some of these conditions I have classified as disorders due to secretional obstruction and/or spasm or paralysis of the larynx I have already stated why I consider spasm much more important than paralysis in discussing poliomyelitis Among these conditions are:

1. *Acute infections and toxins.*
   Bulbar poliomyelitis, tetanus, diphtheria, botulism
2. *Drugs and poisons, especially.*
   Anesthetics and barbiturates.
3. *Central injury*
   Trauma, especially war wounds, tumors, vascular accidents, brain abscess
4. *Systemic disease involving the bulb*

General paresis, tabes, gummata, disseminated sclerosis, amyotrophic lateral sclerosis, syringobulbia, glossolabial paralysis

5. *Myasthenia.*

The primary central injury may be severe and produce coma, swallowing difficulty, fluid accumulation, pulmonary edema, atelectasis and atypical pneumonia. These findings are reported post mortem in many of these conditions, in up to 90% for instance in tetanus (105). Though the primary injury may be serious, it is often self limited, and the outcome may be determined by the depression due to unrecognized anoxia and carbon dioxide accumulation and the interference with respiration and secondary blocking of the lung bed. Even those who hold bulbar poliomyelitis to be often an overwhelming central viral infection must concede that peripheral factors are often vital determinants. The crudest concept of these conditions should provide for clearing the airway and assisting oxygenation and respiration if such impairments are recognized.

Some of the conditions having the same common factor of respiratory obstruction, anoxia and carbon dioxide accumulation, respiratory weakness and the danger from drowning from aspirated secretion will be discussed in more detail.

TETANUS

In 1949 Turner and Galloway (106) reported that tetanus under control by curare presented almost the same picture as bulbar poliomyelitis and responded to the same measures. With the primary focus eliminated this disease usually runs a self limited course of from eight to 12 days. Curare has been used in the treatment of tetanus since 1891 as reported by Cullen (107); tra-

cheotomy had been advised for severe cases of the disease by Spaeth (108); Isaacson and Swanson (109) used a respirator with apparent benefit but without recovery in one case of tetanus, but tracheotomy was not done. We combined the three measures and in our experience the combination was very valuable.

We outlined our treatment of severe tetanus as follows:

1. Drainage and debridement of the focus
2. Antitoxin—although there is doubt about its usefulness except for prophylaxis.
3. Control of convulsions by (a) sedatives and anesthesia, (b) curare
4. Tracheotomy which
   (a) prevents asphyxia from spasm and should be done in all severe cases
   (b) by-passes secretion and prevents aspiration of secretion.
   (c) prevents lung complications such as pulmonary edema, atelectasis and pneumonitis found in 90% of fatal cases.
   (d) allows use of a lower level of curarization.
5 Respirator if needed.
6 Postural drainage and continuous nasal suction if there is any fluid accumulation.
7. Tracheal aspiration
8 Oxygen, usually
9 Parenteral fluids early and gavage feeding later.

Some of these points need further discussion Sedatives in mild cases, especially if supplemented with small doses of curare may control spasm If convulsions at any time threaten to become severe or generalized, tracheotomy, which is easy and safe if done at an early stage, should be performed It is very easy to precipitate a convulsion in which a patient may asphyxiate without tracheotomy.

With tracheotomy one may use a much smaller dosage of curare and thus is much safer. If curare is long used in full enough doses to destroy voluntary muscle movement and tone, there is much danger of a state similar to shock perhaps associated with stasis of circulation We maintain the patients so there are mild tonic movements on using a needle for medication.

A fairly high level of curarization had to be maintained to prevent convulsions. Without mechanical aid respiration was seriously depressed from the drug and also from the action of anoxia and carbon dioxide on the respiratory center Although probably not necessary in all cases, the respirator seemed valuable for exchange of gases as well as for lung movements which probably have much to do with pulmonary circulation and the prevention of atelectasis. Synchronization with the machine may not be perfect, and there may be minor ectopic respiratory movements, but the patient falls rather quickly into rhythm with the respirator and does not wish to be removed until several days after curare has been stopped and normal tone and strength are regained

If tracheotomy has been done a feeding tube may be used relatively early and electrolytes, especially potassium, so given will be taken up in the amount required

The patient may need the respirator up to eight to 10 days and the tracheotomy tube should be left in a few days longer Tracheotomy should be done high to permit care in the respirator

DIPHTHERIA

Diphtheria may produce a complex of laryngeal and deeper obstruction and palatal, pharyngeal and deglutitory paralysis, along with severe toxic depression, as well as anoxia This may have been overlooked in earlier

safety it affords when the condition becomes threatening If, as Von Leden points out, aspiration by catheter or by bronchoscopy affords only temporary relief, tracheotomy is the treatment of choice and should be done at once

BARBITURATE POISONING

In moderately toxic doses of barbiturates, after removing the excess drug from the stomach, the patient sleeps off its effect. With larger doses the effect is a profound depression with slow elimination or detoxification requiring up to five to eight days. Cough is obtunded, secretions accumulate and pulmonary complications develop, or the patients may die of a combination of central drug and anoxic depression with atelectasis and pneumonitis Respiratory efforts may be produced by stimulation with picrotoxin, ephedrine and other drugs, but such management alone is very hazardous Tracheotomy again is a slight step to take to save a life. It bypasses secretions, prevents anoxia by their obstruction and permits their easy removal from the trachea. If respiratory depression is profound and persisting, the respirator should replace less certain methods of artificial respiration. Its use is much safer after tracheotomy. By such techniques some of these patients have recovered from very large doses of the barbiturates

CRANIOCEREBRAL INJURIES

An early experience impressed me with the importance of anoxia in head injury. A patient with sagging tongue, coma and shallow respiration appeared to be dying of shock from a gunshot wound through the frontal lobes On suspicion bronchoscopic aspiration was done and a large mucoid clump was removed. With the patient turned with the face down in moderate postural drain-

age, recovery was fairly prompt In many central injuries anoxia and carbon dioxide accumulation may be an important part of the picture adding markedly to depression and shock

Echols (114) and his group from the Ochsner Clinic state that in patients unconscious from head injuries "the airway is of first importance because of its aid in the prevention of cerebral anoxia, a devastating sequel to craniocerebral trauma" The use of tracheotomy for this purpose has been neglected, but more general interest in this procedure in neurosurgery has followed its employment in tetanus and bulbar poliomyelitis They report that tracheotomy was done in 69 patients solely to prevent or correct respiratory complications. In 57 patients it was done prophylactically.

In head injuries without tracheotomy the nasal passages as a rule are closed by traumatic edema, blood and mucus. The tongue drops back, and hematoma or fracture of the mandible, hyoid or larynx may add to the difficulty Saliva, blood, vomitus and cerebrospinal fluid may pool in the pharynx and run down the trachea The unconscious patients present a grave problem as they are frequently oblivious to secretion, cough little, swallow poorly and breathe slowly with shallow respirations They are likely to have severe anoxia and to develop atelectasis, pneumonitis and pulmonary edema.

The improvement in such patients after tracheotomy, as Echols points out, is usually dramatic Color improves, the patient relaxes, and the labor of breathing diminishes Secretions can easily be removed by suction catheter through the tracheotomy tube "The assurance that the patient will be free of respiratory complications . for the coming desperate days or weeks is a relief to all concerned."

Echols and his group performed tracheotomy in 15 patients with head injuries with gratifying results, although all did not survive. They advise the use of the endotracheal tube if aid is needed for only 12 to 20 hours. If it is to be required for more than 24 hours they urge tracheotomy.

If severe trauma involves the larynx or trachea itself, especially with fracture which may lead to speedy asphyxia by sudden spread of edema or hematoma, tracheotomy should be done prophylactically.

IN NEUROSURGERY

Recently Taylor and Austin (115) recorded the great usefulness of tracheotomy in certain cases in which neurosurgery had been done They point out that in such patients consciousness is diminished perhaps for many days The cough reflex is depressed, liquids or foods may be aspirated into the lungs, the pharyngeal musculature and tongue are relaxed and fluid is likely to reaccumulate after tracheal aspiration Laryngospasm, they believe, may be important. Tracheotomy was in their opinion very valuable in selected neurosurgical patients in preventing anoxia and pulmonary complications They reiterate that "patients in a diminished state of consciousness secondary to organic central nervous system disease may be unduly sensitive to even slight degrees of oxygen want."

The clinician will not infrequently be gratified to find marked and rapid improvement in the neurosurgical patient whose airway has been cleared or on whom a tracheotomy has been done This can be accepted as valid proof that severe symptoms considered due to the central disorder itself should have been ascribed to the overlay of anoxia

If vascular accident, tumor, abscess and the like posi-

tively give such a serious prognosis that nothing could prolong life, intervention would be useless Though this may seem obvious, one cannot always be sure and we have repeatedly seen such patients recover on clearing the airway. The usual procedure is to remove mucus by catheter or bronchoscope sometimes inserting a pharyngeal airway or endotracheal tube If this procedure gives relief with subsequent recurrence of trouble then tracheotomy is done. It does not seem justifiable to let even hopeless patients die of asphyxia, especially if the process is painful and prolonged

In systemic nervous disease involving the bulb, if the condition is not terminal and death soon inevitable, intervention should probably be considered because the effect of anoxic overlay is not easily distinguished from the effects of other medullary damage

In myasthenia gravis treated with neomycin a patient was unconscious and apparently dying with mixed muscle weakness, fluid accumulation and laryngeal spasm. After tracheotomy recovery was dramatic and a fairly normal state of well being was maintained. Others have reported similar cases

Dr Martin Seifert and I surmise that tracheotomy might even be worth a trial in rabies

CONCLUSION

Recently a popular work has explored for us the teeming depths of the sea and shown how the actions taking place affect every other earthly activity, geological and human In a very modest way this essay has attempted to uncover facts commonly ignored or unknown that may have a vital effect in many disease processes Especially has the effort been made to show how easily the peripheral respiratory tract is deranged and yet how far

reaching may be the effect of that disturbance. Respiratory obstruction, anoxia and carbon dioxide accumulation, unless recognized and relieved, perpetuate and augment themselves often to a fatal issue. If recognized and opposed even by relatively simple means they may give some of the most satisfying victories in the field of our endeavor.

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This Book

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By THOMAS C. GALLOWAY, M.D.

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